=> fil reg

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provided by InfoChem.

STRUCTURE FILE UPDATES: 5 MAY 2004 HIGHEST RN 680179-46-8
DICTIONARY FILE UPDATES: 5 MAY 2004 HIGHEST RN 680179-46-8

TSCA INFORMATION NOW CURRENT THROUGH JANUARY 6, 2004

Please note that search-term pricing does apply when
conducting SmartSELECT searches.

Crossover limits have been increased. See HELP CROSSOVER for details.

Experimental and calculated property data are now available. For more
information enter HELP PROP at an arrow prompt in the file or refer
to the file summary sheet on the web at:
http://www.cas.org/ONLINE/DBSS/registryss.html

=> fil zcaplus

FILE 'ZCAPLUS' ENTERED AT 09:00:43 ON 07 MAY 2004
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FILE COVERS 1907 - 7 May 2004 VOL 140 ISS 20
FILE LAST UPDATED: 6 May 2004 (20040506/ED)

This file contains CAS Registry Numbers for easy and accurate
substance identification.

=> fil hcaplus

FILE 'HCAPLUS' ENTERED AT 09:00:46 ON 07 MAY 2004
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FILE COVERS 1907 - 7 May 2004 VOL 140 ISS 20
FILE LAST UPDATED: 6 May 2004 (20040506/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

=> fil biosis

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FILE COVERS 1969 TO DATE.
CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 6 May 2004 (20040506/ED)

FILE RELOADED: 19 October 2003.

=> fil kosmet

FILE 'KOSMET' ENTERED AT 09:00:54 ON 07 MAY 2004
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FILE LAST UPDATED: 06 MAY 2004 <20040506/UP>
FILE COVERS 1968 TO DATE.

>>> SIMULTANEOUS LEFT AND RIGHT TRUNCATION IS AVAILABLE IN THE BASIC INDEX (/BI) FIELD <<<

=> fil stnguide

FILE 'STNGUIDE' ENTERED AT 09:00:59 ON 07 MAY 2004
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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Apr 30, 2004 (20040430/UP).

=> d que l120
L32 (1) SEA FILE=REGISTRY ABB=ON PLU=ON TELOMERASE/CN
L33 (1) SEA FILE=HCAPLUS ABB=ON PLU=ON US2001-893252/AP
L34 SEL PLU=ON L33 1- RN : 27 TERMS
L35 (27) SEA FILE=REGISTRY ABB=ON PLU=ON L34
L36 (12289) SEA FILE=HCAPLUS ABB=ON PLU=ON EXPERIMENTAL CELL RESEARCH/JT

L37 (48) SEA FILE=HCAPLUS ABB=ON PLU=ON L36 AND 252/VL
L38 (1) SEA FILE=HCAPLUS ABB=ON PLU=ON L37 AND (PAGE, T?)/AU
L39 SEL PLU=ON L38 1 RN : 19 TERMS
L40 (19) SEA FILE=REGISTRY ABB=ON PLU=ON L39

L41 (26) SEA FILE=REGISTRY ABB=ON PLU=ON L35 NOT (30516-87-1)/RN
L42 (17) SEA FILE=REGISTRY ABB=ON PLU=ON L40 NOT (120178-12-3 OR 2564-35-4)/RN
L43 (12776) SEA FILE=HCAPLUS ABB=ON PLU=ON L41 OR L42
L44 (4047) SEA FILE=HCAPLUS ABB=ON PLU=ON L32
L45 (864) SEA FILE=HCAPLUS ABB=ON PLU=ON L44 (L) (?HIBIT? OR ?REGU? OR ?BLOCK? OR ?STOP? OR ?HALT? OR ?RUPT? OR ?AGON? OR ?VENT? OR ?REDUC? OR ?IMPED? OR ?SLOW? OR ?HAMPER?)
L46 (3060) SEA FILE=HCAPLUS ABB=ON PLU=ON (?TELOMERASE? (L) (?HIBIT? OR ?REGU? OR ?BLOCK? OR ?STOP? OR ?HALT? OR ?RUPT? OR ?AGON? OR ?VENT? OR ?REDUC? OR ?IMPED? OR ?SLOW? OR ?HAMPER?))
L47 (1) SEA FILE=HCAPLUS ABB=ON PLU=ON (OLIGOMERS/CT) (L) (?TELOMERASE?)
L48 (3) SEA FILE=HCAPLUS ABB=ON PLU=ON (RNA/CT) (L) (?TELOMERASE? (3A) ?OLIGOMER?)
L49 (89) SEA FILE=HCAPLUS ABB=ON PLU=ON (PORPHYRINS/CT) (L) (?CATIONIC?)
L50 (15823) SEA FILE=HCAPLUS ABB=ON PLU=ON L43 OR L48 OR L47 OR L49 OR L45 OR L46
L51 (31500) SEA FILE=HCAPLUS ABB=ON PLU=ON HAIR?/CW
L52 (49003) SEA FILE=HCAPLUS ABB=ON PLU=ON HAIR+PFT,NT,RT/CT
L53 (1894) SEA FILE=HCAPLUS ABB=ON PLU=ON ("HAIR (L) FOLLICLE"/CT OR FOLLICLE/CT OR "FOLLICLE HAIR"/CT OR "HAIR FOLLICLE"/CT)
L54 (744) SEA FILE=HCAPLUS ABB=ON PLU=ON DANDRUFF?/CW
L55 (737) SEA FILE=HCAPLUS ABB=ON PLU=ON (DANDRUFF/CT OR "DANDRUFF SCALP"/CT)
L56 (2332) SEA FILE=HCAPLUS ABB=ON PLU=ON ALOPECIA?/CW
L57 (2433) SEA FILE=HCAPLUS ABB=ON PLU=ON (ALOPECIA/CT OR BALDNESS/CT OR "HAIR LOSS"/CT) OR ("ALOPECIA (L) AREATA"/CT OR "AREATA ALOPECIA"/CT) OR ("ALOPECIA (L) MALE PATTERN"/CT OR "ANDROGENIC ALOPECIA"/CT OR "HEREDITARY ALOPECIA"/CT OR "MALE PATTERN ALOPECIA"/CT OR "MALE PATTERN BALDNESS"/CT)
L58 (404) SEA FILE=HCAPLUS ABB=ON PLU=ON SCALP/CT OR ("SCALP (L) DISEASE"/CT OR "DISEASE SCALP"/CT OR "DISORDER SCALP"/CT OR "SCALP DISEASES"/CT)
L59 (103) SEA FILE=HCAPLUS ABB=ON PLU=ON BALDNESS?/CW
L60 (1712) SEA FILE=HCAPLUS ABB=ON PLU=ON "HAIR PREPARATIONS (L) GROWTH STIMULANTS"/CT OR ("HAIR PREPARATIONS (L) GROWTH STIMULANTS"/CT OR "BALDNESS REMEDIES"/CT OR "GROWTH STIMULANTS HAIR PREPARATIONS"/CT OR "HAIR GROWTH AGENTS"/CT OR "HAIR GROWTH PREPARATIONS"/CT OR "HAIR GROWTH PROMOTERS"/CT OR "HAIR GROWTH STIMULANTS"/CT OR "HAIR TONICS"/CT)
L61 (868) SEA FILE=HCAPLUS ABB=ON PLU=ON (HIRSUTISM/CT OR HYPERTRICHOSIS/CT)
L62 (1) SEA FILE=HCAPLUS ABB=ON PLU=ON HYPERTRICHOSIS/CT
L63 (130085) SEA FILE=HCAPLUS ABB=ON PLU=ON COSMETICS+PFT,NT,RT/CT
L64 (621) SEA FILE=HCAPLUS ABB=ON PLU=ON (DEPILATORIES/CT OR "COSMETICS (L) DEPILATORIES"/CT) OR ("COSMETICS (L) DEPILATORIES"/CT OR DEPILATORIES/CT OR "COSMETIC DEPILATORIES"/CT OR "DEPILATORIES COSMETICS"/CT OR "HAIR REMOVERS"/CT)
L65 (125) SEA FILE=HCAPLUS ABB=ON PLU=ON L50 AND (L51 OR L52 OR L53 OR L54 OR L55 OR L56 OR L57 OR L58 OR L59 OR L60 OR L61 OR L62 OR L63 OR L64)
L66 (9166) SEA FILE=HCAPLUS ABB=ON PLU=ON ((?HAIR?) (5A) (?HIBIT? OR ?REMOV? OR ?REGU? OR ?BLOCK? OR ?STOP? OR ?HALT? OR ?RUPT? OR ?AGON? OR ?VENT? OR ?REDUC? OR ?IMPED? OR ?SLOW? OR ?HAMPER?))
L67 (12) SEA FILE=HCAPLUS ABB=ON PLU=ON L50 AND L66
L68 (128) SEA FILE=HCAPLUS ABB=ON PLU=ON L65 OR L67
L69 (110) SEA FILE=HCAPLUS ABB=ON PLU=ON L68 AND (PY<2002 OR PRY<2002 OR AY<2002)

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L70 (    138597)SEA FILE=HCAPLUS ABB=ON  PLU=ON  (?SHAMPOO? OR ?CONDITIONER?
               OR ?HAIR? OR ?FOLLICLE? OR ?DANDRUFF? OR ?ALOPECIA? OR ?BALD?
               OR ?HIRSUT? OR ?HYPERTRICHO? OR ?DEPILATOR?)
L71 (        37)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L69 AND L70
L72 (    316289)SEA FILE=HCAPLUS ABB=ON  PLU=ON  (?SHAMPOO? OR ?CONDITIONER?
               OR ?BROW? OR ?WHISKER? OR ?HAIR? OR ?FOLLICLE? OR ?DANDRUFF?
               OR ?ALOPECIA? OR ?BALD? OR ?HIRSUT? OR ?HYPERTRICHO? OR
               ?DEPILATOR? OR ?SHAV?)
L73 (        38)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L69 AND L72
L74 (         1)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L73 NOT L71
L75 (        37)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L73 NOT L74
L76          34 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L75 NOT (GENE DELIVERY OR
               ACIDIC GUT OR BIOFILM)/TI
L120         33 SEA FILE=HCAPLUS ABB=ON  PLU=ON  L76 NOT HAIRPIN/IT


=> d que l118
L86 (         1)SEA FILE=REGISTRY ABB=ON  PLU=ON  TELOMERASE/CN
L87 (         1)SEA FILE=HCAPLUS ABB=ON  PLU=ON  US2001-893252/AP
L88             SEL  PLU=ON  L87 1- RN :      27 TERMS
L89 (        27)SEA FILE=REGISTRY ABB=ON  PLU=ON  L88
L90 (     12289)SEA FILE=HCAPLUS ABB=ON  PLU=ON  EXPERIMENTAL CELL RESEARCH/JT

L91 (        48)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L90 AND 252/VL
L92 (         1)SEA FILE=HCAPLUS ABB=ON  PLU=ON  L91 AND (PAGE, T?)/AU
L93             SEL  PLU=ON  L92 1 RN :      19 TERMS
L94 (        19)SEA FILE=REGISTRY ABB=ON  PLU=ON  L93
L95 (        26)SEA FILE=REGISTRY ABB=ON  PLU=ON  L89 NOT (30516-87-1)/RN
L96 (        17)SEA FILE=REGISTRY ABB=ON  PLU=ON  L94 NOT (120178-12-3 OR
               2564-35-4)/RN
L97 (     14458)SEA FILE=BIOSIS ABB=ON  PLU=ON  L95
L98 (         0)SEA FILE=BIOSIS ABB=ON  PLU=ON  L96
L99 (     14458)SEA FILE=BIOSIS ABB=ON  PLU=ON  (L97 OR L98)
L100(      4181)SEA FILE=BIOSIS ABB=ON  PLU=ON  L86
L101(      2706)SEA FILE=BIOSIS ABB=ON  PLU=ON  (?TELOMERASE? (L) (?HIBIT? OR
               ?REGU? OR ?BLOCK? OR ?STOP? OR ?HALT? OR ?RUPT? OR ?AGON? OR
               ?VENT? OR ?REDUC? OR ?IMPED? OR ?SLOW? OR ?HAMPER?))
L102(         6)SEA FILE=BIOSIS ABB=ON  PLU=ON  (L100 (L) (?HIBIT? OR ?REGU?
               OR ?BLOCK? OR ?STOP? OR ?HALT? OR ?RUPT? OR ?AGON? OR ?VENT?
               OR ?REDUC? OR ?IMPED? OR ?SLOW? OR ?HAMPER?))
L103(       226)SEA FILE=BIOSIS ABB=ON  PLU=ON  ?TELOMERASE? (L) ?OLIGO?
L104(      3034)SEA FILE=BIOSIS ABB=ON  PLU=ON  (?PORPHYRIN? OR ?PORPHIN?) (L)
               ?CATION?
L105(    196361)SEA FILE=BIOSIS ABB=ON  PLU=ON  (?SHAMPOO? OR ?CONDITIONER? OR
               ?BROW? OR ?WHISKER? OR ?HAIR? OR ?FOLLICLE? OR ?DANDRUFF? OR
               ?ALOPECIA? OR ?BALD? OR ?HIRSUT? OR ?HYPERTRICHO? OR ?DEPILATOR
               ? OR ?SHAV?)
L106(        66)SEA FILE=BIOSIS ABB=ON  PLU=ON  (L97 OR L98 OR L99 OR L100 OR
               L101 OR L102 OR L103 OR L104) (L) L105
L107(        49)SEA FILE=BIOSIS ABB=ON  PLU=ON  L106 AND (PY<2002 OR MY<2002)
L108(     23714)SEA FILE=BIOSIS ABB=ON  PLU=ON  (?SHAMPOO? OR ?CONDITIONER? OR
               ?BROW? OR ?WHISKER? OR ?HAIR? OR ?FOLLIC? OR ?DANDRUFF? OR
               ?HYPERTRICHO? OR ?ALOPECIA? OR ?BALD? OR ?HIRSUT? OR ?HYPERTRIC
               HO? OR ?DEPILATOR? OR ?SHAV?)/CW,CC,CT
L109(        29)SEA FILE=BIOSIS ABB=ON  PLU=ON  L108 AND (L97 OR L98 OR L99 OR
               L100 OR L101 OR L102 OR L103 OR L104)
L110(        18)SEA FILE=BIOSIS ABB=ON  PLU=ON  L109 AND (PY<2002 OR MY<2002)
L111(        46)SEA FILE=BIOSIS ABB=ON  PLU=ON  L107 NOT L110
L112(        46)SEA FILE=BIOSIS ABB=ON  PLU=ON  L111 NOT HAIRPIN?/TI
L113(        13)SEA FILE=BIOSIS ABB=ON  PLU=ON  L110 NOT (HAIRPIN? OR T CELL?
```

```
                OR POLYOPSIS OR GYNECOMASTIA OR MERKEL)/TI
L114(         24)SEA FILE=BIOSIS ABB=ON  PLU=ON  L112 NOT (CHICKEN OR ?LOOP OR
                HEXAD OR PHOTOFRIN OR CALLI OR GASTRIC OR MALARIA OR METALLOPOR
                PHYRIN OR LIVER OR EGGS OR BOX OR MTERT? OR QUADRUPLEX OR
                HERBICID? OR LYMPH OR COPPER OR CHILEAN OR PAPILLOMAS OR
                COTTON)/TI
L115(         37)SEA FILE=BIOSIS ABB=ON  PLU=ON  (L113 OR L114)
L116(         24)SEA FILE=BIOSIS ABB=ON  PLU=ON  L115 NOT (PROFILE OR OVARIAN
                OR TESTIS OR NEOPLASIA OR HEMATO OR MAMMARY OR POLYPOSIS OR
                THYROID OR RENAL)/TI
L117(         14)SEA FILE=BIOSIS ABB=ON  PLU=ON  L116 NOT (PROTOPORPHYRIN
                IX)/AB
L118          13 SEA FILE=BIOSIS ABB=ON  PLU=ON  L117 NOT (HAIRY CELL LEUKEMIA)/
                IT


=> d que l122
L121(          6)SEA FILE=KOSMET ABB=ON  PLU=ON  ?TELOMERAS? (L) (?HIBIT? OR
                ?REGU? OR ?BLOCK? OR ?STOP? OR ?HALT? OR ?RUPT? OR ?AGON? OR
                ?VENT? OR ?REDUC? OR ?IMPED? OR ?SLOW? OR ?HAMPER?)
L122           3 SEA FILE=KOSMET ABB=ON  PLU=ON  L121 NOT (21042/AN OR 28644/AN
                OR 28703/AN)


=> dup rem l120 l118 l122
DUPLICATE IS NOT AVAILABLE IN 'KOSMET'.
ANSWERS FROM THESE FILES WILL BE CONSIDERED UNIQUE

FILE 'HCAPLUS' ENTERED AT 09:01:33 ON 07 MAY 2004
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FILE 'KOSMET' ENTERED AT 09:01:33 ON 07 MAY 2004
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PROCESSING COMPLETED FOR L120
PROCESSING COMPLETED FOR L118
PROCESSING COMPLETED FOR L122
L123          48 DUP REM L120 L118 L122 (1 DUPLICATE REMOVED)
                ANSWERS '1-33' FROM FILE HCAPLUS
                ANSWERS '34-45' FROM FILE BIOSIS
                ANSWERS '46-48' FROM FILE KOSMET

=> FIL STNGUIDE

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AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Apr 30, 2004 (20040430/UP).

=> d l123 ibib abs hit
YOU HAVE REQUESTED DATA FROM FILE 'BIOSIS, HCAPLUS, KOSMET' - CONTINUE? (Y)/N:y
```

L123 ANSWER 1 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN DUPLICATE 1
ACCESSION NUMBER: 1998:271710 HCAPLUS
DOCUMENT NUMBER: 129:36080
TITLE: Comparative dispositions of ofloxacin in human head, axillary, and public **hairs**
AUTHOR(S): Kosuge, Kazuhiro; Uematsu, Toshihiko; Araki, Sei-Ichi; Matsuno, Hiroyuki; Ohashi, Kyoichi; Nakashima, Mitsuyoshi
CORPORATE SOURCE: Department of Clinical Pharmacology, Hamamatsu University School of Medicine, Hamamatsu, 431-31, Japan
SOURCE: Antimicrobial Agents and Chemotherapy (**1998**), 42(5), 1298-1302
CODEN: AMACCQ; ISSN: 0066-4804
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The distribution of ofloxacin (OFLX) along the shaft of each of three **hair** types, i.e., head, axillary and public, was investigated and compared among five healthy male volunteers 1 to 4 mo after ingestion of OFLX for 1 or 2 days (total dose, 200 or 600 mg). Five strands of each **hair** type were sectioned together into successive 0.5-cm lengths starting from the dermal end, over a length of ≤6 cm, and the OFLX concentration in each **hair** section was measured by high-pressure liquid chromatog. with fluorescence detection. The distribution of OFLX along the head **hair** shaft was narrow, having a single peak even 3 to 4 mo after administration, suggesting a rather uniform growth rate among **hair** strands. The OFLX distribution along axillary or public **hair** shafts tended to be broad, even having two apparent peaks, and the growth rate did not seem uniform. Since axillary **hair** seemed to **stop** growing after having gained a length of ≤4 to 5 cm, it was suggested to enter a resting stage after the growth of ≤3 cm over the 2 to 4 mo after OFLX incorporation. These findings indicate that head **hair** is the most suitable for anal. of individual drug use and the larger growth rate and cycle stage variabilities of strands of the other types of **hair** should be taken into account.

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Comparative dispositions of ofloxacin in human head, axillary, and public **hairs**
SO Antimicrobial Agents and Chemotherapy (**1998**), 42(5), 1298-1302
CODEN: AMACCQ; ISSN: 0066-4804

AB The distribution of ofloxacin (OFLX) along the shaft of each of three **hair** types, i.e., head, axillary and public, was investigated and compared among five healthy male volunteers 1 to 4 mo after ingestion of OFLX for 1 or 2 days (total dose, 200 or 600 mg). Five strands of each **hair** type were sectioned together into successive 0.5-cm lengths starting from the dermal end, over a length of ≤6 cm, and the OFLX concentration in each **hair** section was measured by high-pressure liquid chromatog. with fluorescence detection. The distribution of OFLX along the head **hair** shaft was narrow, having a single peak even 3 to 4 mo after administration, suggesting a rather uniform growth rate among **hair** strands. The OFLX distribution along axillary or public **hair** shafts tended to be broad, even having two apparent peaks, and the growth rate did not seem uniform. Since axillary **hair** seemed to **stop** growing after having gained a length of ≤4 to 5 cm, it was suggested to enter a resting stage after the growth of ≤3 cm over the 2 to 4 mo after OFLX incorporation. These findings

indicate that head **hair** is the most suitable for anal. of individual drug use and the larger growth rate and cycle stage variabilities of strands of the other types of **hair** should be taken into account.

ST ofloxacin disposition head axillary public **hair**

IT **Hair**
Head
Pharmacokinetics
(comparative dispositions of ofloxacin in human head and axillary and public **hairs**)

IT **82419-36-1**, Ofloxacin
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(comparative dispositions of ofloxacin in human head and axillary and public **hairs**)

=> d l123 ibib abs hit 2-33
YOU HAVE REQUESTED DATA FROM FILE 'BIOSIS, HCAPLUS, KOSMET' - CONTINUE? (Y)/N:y

L123 ANSWER 2 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:356593 HCAPLUS
DOCUMENT NUMBER: 138:350819
TITLE: Immortalized mesenchymal cells and its utilization
INVENTOR(S): Hamada, Hirofumi; Kawano, Yutaka; Nakamura, Kiminori; Kobune, Masayoshi; Honmou, Osamu; Tanooka, Atsushi; Oka, Shinichi; Sasaki, Katsunori; Tsuda, Hajime; Ito, Yoshinori; Kato, Junji; Matsunaga, Takuya; Niitsu, Yoshiro
PATENT ASSIGNEE(S): Renomedix Institute Inc.,
SOURCE: PCT Int. Appl., 84 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: Japanese
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLIC
WO 2003038076	A1	20030508	WO 2002-JP11389 20021031 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: JP 2001-335375 A 20011031 <--
AB A method is provided for safely proliferating cord blood-origin hematopoietic stem cells to such an extent as being clin. applicable to, for example, the hematopoietic stem cell transplantation to an adult patient. In order to prepare a large number of mesenchymal stem cells or mesenchymal cells which can be obtained only in an extremely small number by the **conventional** methods, an immortalizing gene such as

telomerase alone is transferred into mesenchymal stem cells, mesenchymal cells or else, and the mesenchymal stem cells thus proliferated are induced into differentiation.

REFERENCE COUNT: 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PRAI JP 2001-335375 A 20011031 <--

AB A method is provided for safely proliferating cord blood-origin hematopoietic stem cells to such an extent as being clin. applicable to, for example, the hematopoietic stem cell transplantation to an adult patient. In order to prepare a large number of mesenchymal stem cells or mesenchymal cells which can be obtained only in an extremely small number by the **conventional** methods, an immortalizing gene such as **telomerase** alone is transferred into mesenchymal stem cells, mesenchymal cells or else, and the mesenchymal stem cells thus proliferated are induced into differentiation.

IT **Hair**
(root; immortalized mesenchymal cells and utilization)

L123 ANSWER 3 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:22648 HCAPLUS
DOCUMENT NUMBER: 138:83416
TITLE: **Telomerase i**
reduction of
INVENTOR(S): Styczynski, P t S.
PATENT ASSIGNEE(S): The Gillette
SOURCE: PCT Int. Appl
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

Applicant

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 2003002077	A2	20030109	WO 2002-US18702	20020612	<--
WO 2003002077	A3	20031016			

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003012755	A1	20030116	US 2001-893252	20010627	<--
EP 1401379	A2	20040331	EP 2002-734785	20020612	<--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRIORITY APPLN. INFO.: US 2001-893252 A1 20010627 <--
WO 2002-US18702 W 20020612

AB Mammalian **hair** growth is **reduced** by applying an **inhibitor** of **telomerase** to the skin.

TI **Telomerase inhibitor** use for **reduction** of **hair** growth

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI WO 2003002077	A2	20030109	WO 2002-US18702	20020612	<--
WO 2003002077	A3	20031016			

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

US 2003012755 A1 20030116 US 2001-893252 20010627 <--
EP 1401379 A2 20040331 EP 2002-734785 20020612 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRAI US 2001-893252 A1 20010627 <--
WO 2002-US18702 W 20020612

AB Mammalian **hair** growth is **reduced** by applying an **inhibitor** of **telomerase** to the skin.

ST **telomerase inhibitor hair** growth **redn**

IT **RNA**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(2'-OMeRNA **telomerase oligomer** and 2'-O-alkyl RNA **telomerase oligomer**; **telomerase inhibitor** for **reduction** of **hair** growth)

IT **Oligomers**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(2'-OMeRNA **telomerase** oligomer and 2'-O-alkyl RNA **telomerase** oligomer; **telomerase inhibitor** for **reduction** of **hair** growth)

IT Androgens
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(androgen-stimulated **hair** growth; **telomerase inhibitor** for **reduction** of **hair** growth)

IT **Porphyrins**
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(**cationic**; **telomerase inhibitor** for **reduction** of **hair** growth)

IT **Hair**
(**follicle**; **telomerase inhibitor** for **reduction** of **hair** growth)

IT **Hair preparations**
(growth **inhibitors**; **telomerase inhibitor** for **reduction** of **hair** growth)

IT **Cosmetics**
Hirsutism
Human
(**telomerase inhibitor** for **reduction** of **hair** growth)

IT mRNA
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**telomerase**; **telomerase inhibitor** for **reduction** of **hair** growth)

IT Telomeres (chromosome)
(telomeric DNA; **telomerase inhibitor** for **reduction** of **hair** growth)

IT DNA
RL: BSU (Biological study, unclassified); BIOL (Biological study)

(telomeric; **telomerase inhibitor** for **redn**
. of **hair** growth)
IT Drug delivery systems
(topical; **telomerase inhibitor** for **reduction**
of **hair** growth)
IT **81-33-4**, 3,4,9,10-Perylenetetracarboxylic diimide
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ligand based on; **telomerase inhibitor** for
reduction of **hair** growth)
IT **128-13-2**, Ursodeoxycholic acid **243-58-3**, 10H-Quindoline
320-67-2, 5-Azacytidine **1393-16-4**, Rubromycin
3056-17-5 25656-92-2 30516-87-1, AZT
38673-65-3 53969-01-0, Purpuromycin **69655-05-6**
, Dideoxyinosine **82419-36-1**, Ofloxacin **88899-62-1**,
Alterperylenol **100986-85-4**, Levofloxacin **117490-04-7**
118353-05-2, , Carbovir **134888-32-7 144245-52-3**
, Fomivirsen **167319-61-1 213416-70-7**
220862-87-3 230287-51-1, Diazaphilonic acid
250256-12-3 482668-83-7 482668-84-8
482668-85-9 482668-86-0
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL
(Biological study); USES (Uses)
(**telomerase inhibitor** for **reduction** of
hair growth)

L123 ANSWER 4 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2003:319267 HCAPLUS
DOCUMENT NUMBER: 138:343858
TITLE: Topical pharmaceuticals for the treatment of inflammatory dermatoses
INVENTOR(S): Maibach, Howard I.; Luo, Eric C.; Hsu, Tsung-Min
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 18 pp., Cont.-in-part of U.S. Ser. No. 972,008.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 25
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
US 2003077301	A1	20030424	US 2002-177250	20020621	<--
US 2001051166	A1	20011213	US 2000-738410	20001214	<--
US 6586000	B2	20030701			
US 2002018803	A1	20020214	US 2000-738395	20001214	<--
US 6719997	B2	20040413			
US 2002034554	A1	20020321	US 2001-972008	20011004	<--
US 6582724	B2	20030624			
ZA 2002004671	A	20030611	ZA 2002-4671	20020611	<--
WO 2004000360	A1	20031231	WO 2003-US19805	20030620	

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC,

NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-465098 B2 19991216 <--
US 2000-569889 A2 20000511 <--
US 2000-607892 B2 20000630 <--
US 2000-738395 A2 20001214 <--
US 2000-738410 A2 20001214 <--
US 2001-972008 A2 20011004 <--
US 2002-177250 A 20020621

OTHER SOURCE(S): MARPAT 138:343858

AB Provided is a topical pharmaceutical composition for the treatment of inflammatory dermatoses, including acne vulgaris, together with methods for its use. The composition and methods involve the topical use of an active agent effective in the treatment of inflammatory dermatoses plus a permeation-enhancing base that gives the composition a pH of 8.0-13.0, preferably 8.0-11.5, and most preferably 8.5-10.5. A topical cream of the invention was prepared by mixing water 370, white petrolatum 250, stearyl alc. 250, propylene glycol 120, sodium lauryl sulfate 10, adapalene 1, methylparaben 0.25, propylparaben 0.15, and KOH 0.01 g.

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	US 2003077301	A1	20030424	US 2002-177250	20020621	<--
	US 2001051166	A1	20011213	US 2000-738410	20001214	<--
	US 6586000	B2	20030701			
	US 2002018803	A1	20020214	US 2000-738395	20001214	<--
	US 6719997	B2	20040413			
	US 2002034554	A1	20020321	US 2001-972008	20011004	<--
	US 6582724	B2	20030624			
	ZA 2002004671	A	20030611	ZA 2002-4671	20020611	<--
	WO 2004000360	A1	20031231	WO 2003-US19805	20030620	

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI	US 1999-465098	B2	19991216	<--
	US 2000-569889	A2	20000511	<--
	US 2000-607892	B2	20000630	<--
	US 2000-738395	A2	20001214	<--
	US 2000-738410	A2	20001214	<--
	US 2001-972008	A2	20011004	<--
	US 2002-177250	A	20020621	

IT **Hair**
(**follicle**, folliculitis; topical pharmaceuticals for treatment of inflammatory dermatoses)

IT 57-13-6, Urea, biological studies 60-54-8, Tetracycline 64-04-0, Benzeneethanamine 68-12-2, N,N-Dimethyl formamide, biological studies 69-72-7, Salicylic acid, biological studies 78-96-6, Isopropanolamine 94-36-0, Benzoyl peroxide., biological studies 96-45-7, Ethylene thiourea 102-71-6, Triethanolamine, biological studies 108-27-0, 5-Methyl-2-pyrrolidone 108-46-3, Resorcinol, biological studies 111-42-2, Diethanolamine, biological studies 114-07-8, Erythromycin 120-89-8, Oxalylurea 121-44-8, Triethylamine, biological studies 122-20-3, Triisopropanolamine 123-99-9, Azelaic acid, biological studies

124-22-1, Dodecylamine 124-30-1, Stearylamine 124-94-7, Triamcinolone 127-19-5, N,N-Dimethyl acetamide 127-56-0, Sodium sulfacetamide 134-62-3, Diethyl-m-toluamide 302-79-4, Tretinoin 461-72-3, Hydantoin 564-25-0, Doxycycline 616-45-5, 2-Pyrrolidone 872-50-4, 1-Methyl-2-pyrrolidone, biological studies 1118-92-9 1305-62-0, Calcium hydroxide, biological studies 1309-42-8, Magnesium hydroxide 1310-58-3, Potassium hydroxide, biological studies 1310-73-2, Sodium hydroxide, biological studies 1336-21-6, Ammonium hydroxide 1643-20-5 2687-91-4, 1-Ethyl-2-pyrrolidone 2915-94-8 5075-92-3, 1,5-Dimethyl-2-pyrrolidone 5809-41-6 5917-47-5, N-Dodecylpiperidine 6935-65-5, Dimethyl-m-toluamide 7704-34-9, Sulfur, biological studies 10118-90-8, Minocycline 13127-82-7 14433-76-2, N,N-Dimethyldecanamide 15416-74-7, Dodecylpyridinium 15686-71-2, Cephalexin 16528-77-1, N-Octadecyl morpholine 16613-87-9, N-Dodecylethanolamine 18323-44-9, Clindamycin 18494-58-1 18494-60-5 20257-67-4 20422-09-7 26027-37-2 28602-31-5, Toluamide 35902-61-5, N-(2-Methoxyethyl)dodecylamine 35924-17-5 39236-46-9, Imidazolidinyl urea 52665-42-6 55972-87-7, Tributanol amine 57654-76-9 59227-89-3, 1-Dodecylazacycloheptan-2-one 70974-50-4 72816-70-7 79448-06-9, Dibutanol amine **82419-36-1**, Ofloxacin 106685-40-9, Adapalene 118292-40-3, Tazarotene 124858-35-1, Nadifloxacin 137909-16-1, 1-Propyl-3-dodecylpyrrolidine 161695-39-2 161695-40-5
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(topical pharmaceuticals for treatment of inflammatory dermatoses)

L123 ANSWER 5 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:555632 HCAPLUS
DOCUMENT NUMBER: 137:106068
TITLE: Pluripotent adult stem cells derived from regenerative tissue
INVENTOR(S): Soria Escoms, Bernat; Be ig
Macia, Juan Antonio; Mar nat
Wasser, Roberto
PATENT ASSIGNEE(S): Cardion A.-G., Germany
SOURCE: PCT Int. Appl., 40 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002057430	A2	20020725	WO 2002-EP475	20020118 <--
WO 2002057430	A3	20031016		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

EP 1370642	A2	20031217	EP 2002-712824	20020118 <--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR

PRIORITY APPLN. INFO.:	EP 2001-101333	A	20010120 <--
	US 2001-287105P	P	20010427 <--

US 2001-324008P P 20010921 <--
WO 2002-EP475 W 20020118

AB The **invention** concerns a pluripotent adult stem cell population derived from regenerative tissue, having alkaline phosphatase activity, high levels of **telomerase** activity and the ability to form derivs. of all three embryonic germ layers and/or the ability to form embryoid bodies. An object of the present **invention** is to provide isolated pluripotent adult stem cell and progenitor cell populations, derived from regenerative tissue, which can differentiate into any cell type, and methods for isolating and enriching pluripotent adult stem cell and progenitor cell populations.

PRAI EP 2001-101333 A 20010120 <--
US 2001-287105P P 20010427 <--
US 2001-324008P P 20010921 <--
WO 2002-EP475 W 20020118

AB The **invention** concerns a pluripotent adult stem cell population derived from regenerative tissue, having alkaline phosphatase activity, high levels of **telomerase** activity and the ability to form derivs. of all three embryonic germ layers and/or the ability to form embryoid bodies. An object of the present **invention** is to provide isolated pluripotent adult stem cell and progenitor cell populations, derived from regenerative tissue, which can differentiate into any cell type, and methods for isolating and enriching pluripotent adult stem cell and progenitor cell populations.

IT **Hair**
(papilla; pluripotent adult stem cells derived from regenerative tissue)

L123 ANSWER 6 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:314743 HCAPLUS
DOCUMENT NUMBER: 136:345786
TITLE: Sustained release delivery system containing an aqueous bicellar matrix containing a phospholipid
INVENTOR(S): Kestel, Frederic Amnon
PATENT ASSIGNEE(S): Advanced Delivery Systems Aps, Den.
SOURCE: PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002032395	A2	20020425	WO 2001-IL966	20011018 <--
WO 2002032395	A3	20021219		

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

AU 2002010894	A5	20020429	AU 2002-10894		20011018 <--

PRIORITY APPLN. INFO.: IL 2000-139177 A 20001020 <--
WO 2001-IL966 W 20011018 <--

AB The invention relates to a sustained release delivery system for the delivery of an active agent to a warm-blooded animal and to uses thereof.

The delivery system comprises an aqueous bicellar matrix that is liquid at temps. below ambient temperature and forms a biodegradable gel at body temperature of said animal and an active agent, and optionally further comprises pharmaceutically acceptable additive, carrier and/or diluent. The aqueous bicellar matrix is preferably a mixture of a lipid, preferably phospholipid, and a detergent in water. The sustained release of toluidine blue was determined from a bicellar phase containing HMPC and DHPC (dihyexanoylphosphatidylcholine).

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 2002032395	A2	20020425	WO 2001-IL966	20011018	<--
	WO 2002032395	A3	20021219			
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG					
	AU 2002010894	A5	20020429	AU 2002-10894	20011018	<--
PRAI	IL 2000-139177	A	20001020	<--		
	WO 2001-IL966	W	20011018	<--		

IT **Cosmetics**
(**depilatories**; sustained release delivery system containing an aqueous bicellar matrix containing a phospholipid)

IT 50-02-2, Dexamethasone 50-23-7, Hydrocortisone 50-24-8, Prednisolone 50-56-6, Oxytocin, biological studies 50-76-0, Actinomycin d 50-78-2, Aspirin 51-17-2, Benzimidazole 52-28-8, Codeine phosphate 53-06-5, Cortisone 53-86-1, Indomethacin 54-42-2, Idoxuridine 56-75-7, Chloramphenicol 57-42-1, Meperidine 57-62-5, Chlortetracycline 57-92-1, Streptomycin, biological studies 58-82-2, Bradykinin 59-05-2, Methotrexate 59-87-0, Nitrofurazone 60-54-8, Tetracycline 62-44-2, Phenacetin 64-31-3, Morphine sulfate 65-45-2, Salicylamide 65-49-6, p-Aminosalicylic acid 69-72-7, Salicylic acid, biological studies 70-00-8, Trifluridine 76-22-2, Camphor 76-42-6, Oxycodone 85-79-0, Dibucaine 87-28-5, Glycol salicylate 89-78-1, Menthol 91-22-5, Quinoline, biological studies 93-60-7, Methyl nicotinate 94-09-7, Benzocaine 103-90-2, Acetaminophen 108-95-2, Phenol, biological studies 112-38-9, Undecylenic acid 114-07-8, Erythromycin 119-36-8, Methyl salicylate 124-94-7, Triamcinolone 137-58-6, Lidocaine 143-71-5, Hydrocodone bitartrate 148-79-8, Thiabendazole 152-97-6, Fluocortolone 154-93-8, Bcnu 359-83-1, Pentazocine 378-44-9, Betamethasone 389-08-2, Nalidixic acid 466-99-9, Hydromorphone 469-62-5, Propoxyphene 552-94-3, Salsalate 557-08-4, Zinc undecylenate 768-94-5, Amantadine 1066-17-7, Colistin 1393-25-5, Secretin 1400-61-9, Nystatin 1403-66-3, Gentamicin 1404-00-8, Mitomycin 1404-04-2, Neomycin 1405-87-4, Bacitracin 1405-97-6, Gramicidin 1406-05-9, Penicillin 1406-11-7, Polymyxin 1407-47-2, Angiotensin 1639-60-7, Propoxyphene hydrochloride 1947-37-1, Tetragastrin 2174-16-5 2398-96-1, Tolnaftate 3546-41-6, Molevac 5534-95-2, Pentagastrin 5536-17-4, Vidarabine 7439-88-5, Iridium, biological studies 7440-14-4, Radium, biological studies 7440-46-2, Cesium, biological studies 7440-65-5, Yttrium, biological studies 7553-56-2, Iodine, biological studies 7681-93-8, Natamycin 7689-03-4, Camptothecin 8011-61-8, Tyrocidine 9002-60-2, Adrenocorticotropic hormone, biological studies 9002-62-4, Prolactin, biological studies 9002-72-6, Somatotropin 9002-76-0, Gastrin 9004-10-8, Insulin,

biological studies 9007-12-9, Calcitonin 9007-92-5, Glucagon, biological studies 9015-94-5, Renin, biological studies 9025-39-2, Heparinase 9034-39-3, Somatoliberin 9034-40-6, Luliberin 9061-61-4, Nerve growth factor 11000-17-2, Vasopressin 11056-06-7, Bleomycin 11111-12-9, Cephalosporin 12629-01-5, Human growth hormone 12633-72-6, Amphotericin 15687-27-1, Ibuprofen 18323-44-9, Clindamycin 20830-81-3, Daunorubicin 22494-42-4, Diflunisal 22916-47-8, Miconazole 23214-92-8, Doxorubicin 23593-75-1, Clotrimazole 24305-27-9, TRH 25377-66-6, Pyrimidine, tetrahydro- 27203-92-5, Tramadol 27220-47-9, Econazole 32986-56-4, Tobramycin 33069-62-4, Paclitaxel 35523-45-6, Fludalanine 38194-50-2, Sulindac 41575-94-4, Carboplatin 41621-49-2, Ciclopirox olamine 55694-83-2, Pentizidone 59277-89-3, Acyclovir 60118-07-2, Endorphin 62229-50-9, Egf 65277-42-1, Ketoconazole 65472-88-0, Naftifine 66419-50-9, Bovine growth hormone 70288-86-7, Ivermectin 79217-60-0, Cyclosporin 81627-83-0, M-CSF 82410-32-0, Ganciclovir **82419-36-1**, Ofloxacin 83869-56-1, GM-CSF 86386-73-4, Fluconazole 89213-87-6, Atrial natriuretic peptide-28 (human) 96352-57-7, Glucagon-like peptide 101828-21-1, Butenafine 104227-87-4, Famciclovir 114977-28-5, Taxotere 124832-26-4, Valacyclovir 126467-48-9, Porcine growth hormone 143011-72-7, G-CSF 149659-36-9, Hylak forte 196618-13-0, Oseltamivir 404346-18-5, Symbioflor1 416841-09-3, Galivert 416841-10-6, Heralvent 416841-11-7, Oricant 416841-17-3, Mucokehl 416841-18-4, Mutaflor 416841-19-5, Paidoflor 416841-20-8, Omnifloran 416841-22-0, Pefrakehl 416841-23-1, Prosymbioflor 416841-43-5, Symbioflor 2 416841-44-6, Taheebo 416841-45-7, Trenev trio

RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(sustained release delivery system containing an aqueous bicellar matrix containing a phospholipid)

L123 ANSWER 7 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:717056 HCAPLUS
DOCUMENT NUMBER: 137:226655
TITLE: Methods for treatment of neuro- and nephro- disorders and therapeutic toxicities using aminothiol compounds
INVENTOR(S): Stogniew, Martin; Alberts, David S.; Kaplan, Edward H.
PATENT ASSIGNEE(S): U.S. Bioscience, Inc., USA
SOURCE: U.S. Pat. Appl. Publ., 15 pp., Division of U. S. Ser. No. 429,290.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 2002132795	A1	20020919	US 2002-137686	20020503 <--
US 6586476	B1	20030701	US 1999-429290	19991028 <--

PRIORITY APPLN. INFO.: US 1999-429290 A3 19991028 <--
US 1997-987550 A3 19971209 <--
OTHER SOURCE(S): MARPAT 137:226655

AB The present invention relates to new uses of S-2-(3-aminopropylamino)ethyl dihydrogen phosphorothioate (amifostine) and other aminothiol compds. to treat and reverse toxicities caused by therapeutic agents, radiation treatment or diabetes. In particular, the invention provides a method for treating neurotoxicity and nephrotoxicity associated with the administration of chemotherapeutic agents. Cancer patients with neurotoxicities from chemotherapy treatment were treated with amifostine.

	PATENT NO.	KIND	DATE		APPLICATION NO.	DATE
PI	US 2002132795	A1	20020919		US 2002-137686	20020503 <--
	US 6586476	B1	20030701		US 1999-429290	19991028 <--
PRAI	US 1999-429290	A3	19991028	<--		
	US 1997-987550	A3	19971209	<--		

IT **Alopecia**
Cytoprotective agents
Human
Kidney, disease
Mammalia
Nerve, disease
Toxicants
Toxicity
(treatment of neuro- and nephro- disorders and therapeutic toxicities using aminothiol compds.)

IT 57-22-7, Vincristine 865-21-4, Vinblastine 1397-89-3, Amphotericin B 1403-66-3, Gentamicin 1404-90-6, Vancomycin **3056-17-5**, Stavudine 7481-89-2, Zalcitabine 8063-07-8, Kanamycin 15663-27-1, Cisplatin 20830-81-3, Daunorubicin 23214-92-8, Doxorubicin 30516-87-1, 3'-Azido-3'-deoxythymidine 32986-56-4, Tobramicin 33069-62-4, Paclitaxel 33419-42-0, Etoposide 37517-28-5, Amikacin 41575-94-4, Carboplatin **69655-05-6**, Didanosine 95058-81-4, Gemcitabine 114977-28-5, Docetaxel 125317-39-7, Navelbine 134678-17-4, Lamivudine
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(toxicity from; treatment of neuro- and nephro- disorders and therapeutic toxicities using aminothiol compds.)

L123 ANSWER 8 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:553061 HCAPLUS
DOCUMENT NUMBER: 137:103928
TITLE: Use of alkanoyloxymethyl esters for inhibiting histone deacetylase and treatment of cancer and other diseases
INVENTOR(S): Lan-Hargest, Hsuan-Yin; Wiech, Norbert L.
PATENT ASSIGNEE(S): Beacon Laboratories, Inc., USA
SOURCE: Eur. Pat. Appl., 14 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1224931	A1	20020724	EP 2001-310689	20011220 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 2002143055	A1	20021003	US 2000-742729	20001221 <--
US 6693132	B2	20040217		

PRIORITY APPLN. INFO.: US 2000-742729 A 20001221 <--
AB The use of propionoyloxymethyl propionate and butyroyloxymethyl butyrate (preparation described) in treating illness is disclosed. Treatable illnesses, include cancer, hematol. disorders and inherited metabolic disorders, as well as other conditions. The compds. are effective in the inhibition of histone deacetylase.
REFERENCE COUNT: 7 THERE ARE 7 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI	EP 1224931	A1	20020724	EP 2001-310689	20011220 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
	US 2002143055	A1	20021003	US 2000-742729	20001221 <--
	US 6693132	B2	20040217		
PRAI	US 2000-742729	A	20001221 <--		

IT **Hair**
(**follicle**, protection against injury to; alkanoyloxymethyl esters for inhibiting histone deacetylase and treatment of cancer and other diseases)

IT 9076-57-7, Histone deacetylase **120178-12-3**, **Telomerase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**inhibition**; alkanoyloxymethyl esters for **inhibiting** histone deacetylase and treatment of cancer and other diseases)

L123 ANSWER 9 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2002:486177 HCAPLUS
DOCUMENT NUMBER: 137:47012
TITLE: Preparation of δ-dicarbonyl compounds as inhibitors of histone deacetylase.
INVENTOR(S): Lan-Hargest, Hsuan-Yin; Wiech, Norbert L.
PATENT ASSIGNEE(S): Beacon Laboratories, Inc., USA
SOURCE: Eur. Pat. Appl., 26 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 1216986	A2	20020626	EP 2001-310693	20011220 <--
EP 1216986	A3	20021204		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 6562995	B1	20030513	US 2000-742588	20001221 <--
US 2002137775	A1	20020926	US 2001-858948	20010517 <--
US 6667341	B2	20031223		
US 2003171409	A1	20030911	US 2002-282255	20021029 <--
PRIORITY APPLN. INFO.:			US 2000-742588 A	20001221 <--
			US 2001-858948 A1	20010517 <--

OTHER SOURCE(S): MARPAT 137:47012
AB R1COXCH2YCR2 [X = O, S, NR; Y = S, NR, CH2; R = H, Me; R1, R2 = (CH2)o(R3)p(CH2)q(R4)r(CH2)sZ; R3, R4 = CH:CH, C.tplbond.C, S, O; Z = H, (substituted) aryl, heteroaryl, cycloalkyl, alkoxy; o, p, q, r, s = 0-10], were prepared Thus, N-methylbutyramide (preparation given) in THF/Me2SO at 0-5° was treated with NaH then with chloromethyl cinnamate in THF followed by stirring overnight at room temperature to give 41.8% N-methylbutyramidomethyl cinnamate. The latter inhibited proliferation of PC-3 prostate cancer cells with IC50 = 100 μM.

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 1216986	A2	20020626	EP 2001-310693	20011220 <--
EP 1216986	A3	20021204		
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR				
US 6562995	B1	20030513	US 2000-742588	20001221 <--
US 2002137775	A1	20020926	US 2001-858948	20010517 <--
US 6667341	B2	20031223		

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     US 2003171409     A1   20030911       US 2002-282255   20021029 <--
PRAI US 2000-742588    A    20001221  <--
     US 2001-858948    A1   20010517  <--
IT   Hair
       (follicle, protectants; preparation of δ-dicarbonyl compds.
       as inhibitors of histone deacetylase)
IT   9076-57-7, Histone deacetylase 120178-12-3, Telomerase
     RL: BSU (Biological study, unclassified); BIOL (Biological study)
       (inhibitors; preparation of δ-dicarbonyl compds. as
       inhibitors of histone deacetylase)

L123 ANSWER 10 OF 48  HCAPLUS  COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER:        2002:486175  HCAPLUS
DOCUMENT NUMBER:         137:63074
TITLE:                   Preparation of acetyloxymethyl esters and their
                         therapeutic applications
INVENTOR(S):             Lan-Hargest, Hsuan-Yin; Weich, Norbert L.
PATENT ASSIGNEE(S):      Beacon Laboratories, Inc., USA
SOURCE:                  Eur. Pat. Appl., 30 pp.
                         CODEN: EPXXDW
DOCUMENT TYPE:           Patent
LANGUAGE:                English
FAMILY ACC. NUM. COUNT:  1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ---------------   ----  --------      ---------------  --------
     EP 1216984         A1   20020626      EP 2001-310692   20011220 <--
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
     US 2002161045      A1   20021031      US 2000-742727   20001221 <--
     US 6720445         B2   20040413
     US 2002119996      A1   20020829      US 2002-55898    20020128 <--
     US 6699902         B2   20040302
PRIORITY APPLN. INFO.:                  US 2000-742727   A  20001221 <--
OTHER SOURCE(S):         MARPAT 137:63074
AB   Novel acetyloxymethyl esters, RCOOCH2OCOMe [I; R = (un)substituted
     alkenyl, (un)substituted alkynyl, a cis or trans retinoyl,
     Z(X)o-(R1)p-(R2)q; Z = H, (un)substituted aryl, heteroaryl, cycloalkyl,
     alkoxy; n = 3, >3; X = S, O, CO, CH2; R1 = S, O, CH:CH, C.tplbond.C; R2 =
     CH2, CH:CH, C.tplbond.C; o, p, q = same or different each between 0-10,
     but when o = 0 and R1 or R2 = CH:CH or C.tplbond.C, Z is not H or alkoxy],
     were prepd for treating an illness, including cancer, hemol. disorders and
     inherited metabolic disorders, and treating or ameliorating other
     conditions.  I are effective in the inhibition of histone deacetylase.
     Thus, cinnamoyloxymethyl acetate (II) was prepared by the reaction of
     cinnamic acid and chloromethyl acetate.  II showed IC50 = 12.5μM
     against PC-3 prostate breast cancer cells.
REFERENCE COUNT:         24    THERE ARE 24 CITED REFERENCES AVAILABLE FOR THIS
                               RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ---------------   ----  --------      ---------------  --------
PI   EP 1216984         A1   20020626      EP 2001-310692   20011220 <--
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, SI, LT, LV, FI, RO, MK, CY, AL, TR
     US 2002161045      A1   20021031      US 2000-742727   20001221 <--
     US 6720445         B2   20040413
     US 2002119996      A1   20020829      US 2002-55898    20020128 <--
     US 6699902         B2   20040302
PRAI US 2000-742727    A    20001221  <--
```

IT **Hair**
(**follicle**, injury treatment; preparation of acetyloxymethyl esters as antitumor agents)
IT **120178-12-3, Telomerase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(activity **inhibition**; preparation of acetyloxymethyl esters as antitumor agents)

L123 ANSWER 11 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2001:525887 HCAPLUS
DOCUMENT NUMBER: 135:127191
TITLE: Pharmaceutical and cosmetic carrier or composition for topical application containing a fatty acid, a fatty alcohol and an oil
INVENTOR(S): Eini, Meir; Tamarkin, Dov
PATENT ASSIGNEE(S): Thixo Ltd., Israel
SOURCE: PCT Int. Appl., 76 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 2001051014	A1	20010719	WO 2001-IL25	20010110	<--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG					
US 6348229	B1	20020219	US 2000-526509	20000316	<--
EP 1250116	A1	20021023	EP 2001-900239	20010110	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
JP 2003528821	T2	20030930	JP 2001-551438	20010110	<--
US 2003157138	A1	20030821	US 2003-392071	20030319	<--

PRIORITY APPLN. INFO.:

IL 2000-133968	A	20000110	<--
IL 2000-133969	A	20000110	<--
US 2000-526509	A	20000316	<--
IL 2000-137051	A	20000627	<--
IL 2000-137052	A	20000627	<--
US 2000-216162P	P	20000703	<--
US 2000-653267	A	20000831	<--
WO 2001-IL25	W	20010110	<--

AB A pharmaceutical or cosmetic carrier or composition for topical application, characterized by rheol. properties which render the carrier or composition semi-solid at rest and a liquid upon application of shear forces, is described. The composition or carrier are prepared by mixing (by weight) 1-25% of a solidifying agent, such as a long-chain fatty alc. and a fatty acid, and 75-99% of a hydrophobic solvent, such as an animal, mineral, silicone, or plant-derived oil, wherein at least one of them has therapeutic or cosmetic benefits, in the presence or absence of a biol. active substance. For example, behenic acid (10 g) was heated to 80° and mixed with light paraffin oil (90 g) preheated to the same temperature Then glycerin (10

g), tristearin (10 g), and an antioxidant mixture (1 g) were added by agitation. Bifunazole (1.2 g) and diflucortolone valerate (0.12 g) were added and the mixture was poured into containers (5 g tubes) and was allowed to cool spontaneously. While the mixture cooled to ambient temperature it gradually turned into a semisolid, i.e., an ointment containing the antifungal agent.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PI WO 2001051014 A1 **20010719**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI WO 2001051014	A1	20010719	WO 2001-IL25	20010110	<--
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG					
US 6348229	B1	20020219	US 2000-526509	20000316	<--
EP 1250116	A1	20021023	EP 2001-900239	20010110	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR					
JP 2003528821	T2	20030930	JP 2001-551438	20010110	<--
US 2003157138	A1	20030821	US 2003-392071	20030319	<--

PRAI IL 2000-133968	A	20000110	<--
IL 2000-133969	A	20000110	<--
US 2000-526509	A	20000316	<--
IL 2000-137051	A	20000627	<--
IL 2000-137052	A	20000627	<--
US 2000-216162P	P	20000703	<--
US 2000-653267	A	20000831	<--
WO 2001-IL25	W	20010110	<--

IT **Cosmetics**
(**depilatories**; topical compns. containing fatty acid, fatty alc. and oil for pharmaceutical and cosmetic uses)

IT **Hair preparations**
(**growth stimulants**; topical compns. containing fatty acid, fatty alc. and oil for pharmaceutical and cosmetic uses)

IT Acne
Antibacterial agents
Antibiotics
Antihistamines
Antiulcer agents
Antiviral agents
Autoimmune disease
Cosmetics
Eczema
Erythema
Fungicides
Immunosuppressants
Mucous membrane
Psoriasis
Seborrhea
Skin preparations (pharmaceutical)
Wound healing promoters
(topical compns. containing fatty acid, fatty alc. and oil for pharmaceutical and cosmetic uses)

IT 50-23-7, Hydrocortisone 56-75-7, Chloramphenicol 60-54-8, Tetracycline 67-73-2, Fluocinolone acetonide 76-25-5, Triamcinolone acetonide 94-36-0, Benzoyl peroxide, biological studies 98-92-0, vitamin B3 106-14-9, 12-Hydroxystearic acid 114-07-8, Erythromycin 118-74-1, Hexachlorobenzene 120-51-4, Benzyl benzoate 121-75-5, Malathion 126-07-8, Griseofulvin 302-79-4, Tretinoin 483-63-6, Crotamiton 768-94-5, Amantadine 1229-29-4, Doxepine hydrochloride 1397-89-3, Amphotericin B 1406-05-9, Penicillin 2022-85-7, Flucytosine 2152-44-5, Betamethasone valerate **3056-17-5**, Stavudine 3093-35-4, Halcinonide 4759-48-2, Isotretinoin 5536-17-4, Vidarabine 5593-20-4, Betamethasone dipropionate 7681-11-0, Potassium iodide, biological studies 12650-69-0, Mupirocin 13392-28-4, Rimantadine 18323-44-9, Clindamycin 22916-47-8, Miconazole 23593-75-1, Clotrimazole 25122-46-7, Clobetasol propionate 29342-05-0, Ciclopirox 30516-87-1, Zidovudine 36791-04-5, Ribavirin 57524-89-7, Hydrocortisone valerate 59198-70-8, Diflucortolone valerate 59277-89-3, Acyclovir 60628-96-8, Bifonazole 65277-42-1, Ketoconazole 66852-54-8, Halobetasol propionate 78613-35-1, Amorolfine 79217-60-0, Cyclosporin 82410-32-0, Gancyclovir 84625-61-6, Itraconazole 86386-73-4, Fluconazole 91161-71-6, Terbinafine 106685-40-9, Adapalene 108436-80-2, Rociclovir 127779-20-8, Saquinavir 129618-40-2, Nevirapine 134678-17-4, Lamivudine 136817-59-9, Delavirdine 150378-17-9, Indinavir 155213-67-5, Ritonavir 159989-64-7, Nelfinavir
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(topical compns. containing fatty acid, fatty alc. and oil for pharmaceutical and cosmetic uses)

L123 ANSWER 12 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:880923 HCAPLUS
DOCUMENT NUMBER: 134:37055
TITLE: Methods and compositions using FGF inhibitors and agonists for modulating cell proliferation and cell death
INVENTOR(S): Au, Jessie L. S.; Wientjes, M. Guillaume
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 143 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000074634	A2	20001214	WO 2000-US40103	20000605 <--
WO 2000074634	C2	20020926		
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1206234	A2	20020522	EP 2000-943429	20000605 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL				
JP 2003503313	T2	20030128	JP 2001-501171	20000605 <--
US 6599912	B1	20030729	US 2000-587559	20000605 <--
US 2004010001	A1	20040115	US 2003-464018	20030618 <--

PRIORITY APPLN. INFO.: US 1999-137345P P 19990603 <--
US 1999-165983P P 19991117 <--
US 1999-172031P P 19991223 <--
US 2000-187445P P 20000307 <--
US 2000-587559 A3 20000605 <--
WO 2000-US40103 W 20000605 <--

AB Methods and compns. for modulating the FGF effect on the sensitivity of malignant and normal cells to anticancer agents are provided. In particular, methods and compns. for inhibiting FGF-induced resistance to a broad spectrum of anticancer agents in solid and soft-tissue tumors, metastatic lesions, leukemia and lymphoma are provided. Preferably, the compns. include at least one FGF inhibitor in combination with a cytotoxic agents, e.g., antimicrotubule agents, topoisomerase I inhibitors, topoisomerase II inhibitors, antimetabolites, mitotic inhibitors, alkylating agents, intercalating agents, agents capable of interfering with a signal transduction pathway (e.g., g., a protein kinase C inhibitor, e.g., an anti-hormone, e.g., an antibody against growth factor receptors), an agent that promote apoptosis and/or necrosis, an interferon, an interleukin, a tumor necrosis factor, and radiation. In other embodiments, methods and composition for protecting a cell in a subject, from one or more of killing, inhibition of growth or division or other damage caused, e.g., by a cytotoxic agent, are provided. Preferably, the method includes administering to the subject an effective amount of at least one FGF agonist, thereby treating the cell, e.g., protecting or reducing the damage to the dividing cell from said cytotoxic agent. FGF gene expression-based methods for diagnosis of proliferative disorders are also disclosed.

PI WO 2000074634 A2 **20001214**

	PATENT NO.	KIND	DATE		APPLICATION NO.	DATE	
PI	WO 2000074634	A2	20001214		WO 2000-US40103	20000605	<--
	WO 2000074634	C2	20020926				
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK						
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG						
	EP 1206234	A2	20020522		EP 2000-943429	20000605	<--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL						
	JP 2003503313	T2	20030128		JP 2001-501171	20000605	<--
	US 6599912	B1	20030729		US 2000-587559	20000605	<--
	US 2004010001	A1	20040115		US 2003-464018	20030618	<--
PRAI	US 1999-137345P	P	19990603	<--			
	US 1999-165983P	P	19991117	<--			
	US 1999-172031P	P	19991223	<--			
	US 2000-187445P	P	20000307	<--			
	US 2000-587559	A3	20000605	<--			
	WO 2000-US40103	W	20000605	<--			

IT **Hair**
(**follicle**; GF **inhibitors** and **agonists** for modulating cell proliferation and cell death)

IT 50-07-7, Mitomycin C 50-18-0, Cyclophosphamide 50-44-2, 6-Mercaptopurine 50-91-9, 5-Fluorodeoxyuridine 52-24-4, Thiotepa 54-91-1, Pipobroman 55-86-7, Nitrogen mustard 55-98-1, Busulfan 57-22-7, Vincristine 58-61-7, Adenosine, biological studies 59-05-2, Methotrexate 66-75-1, Uracil mustard 147-94-4, Cytarabine 148-82-3,

Melphalan 154-42-7, 6-Thioguanine 154-93-8, BCNU 305-03-3, Chlorambucil 316-46-1, 5-Fluorouridine **320-67-2**, 5-Azacytidine 865-21-4, Vinblastine 2353-33-5, 5-Aza-2'-deoxycytidine 3778-73-2, Ifosfamide 4291-63-8, Cladribine 4342-03-4, Dacarbazine 5854-93-3, Alanosine 7689-03-4, Camptothecin 18378-89-7, Plicamycin 20830-81-3, Daunorubicin 29767-20-2, Teniposide 30868-30-5, Pyrazofurin 32954-58-8, 4-Ipomeanol 33419-42-0, Etoposide 38077-12-2, NSC 343513 42228-92-2, Acivicin 51264-14-3, Amsacrine 51321-79-0, PALA 52128-35-5, Trimetrexate 53643-48-4, Vindesine 53910-25-1, Pentostatin 56605-16-4, Spiromustine 58957-92-9, Idarubicin 60084-10-8, Tiazofurin 65271-80-9, Mitoxantrone 71486-22-1, Vinorelbine 97534-21-9, Merbarone 105118-12-5, Piroxantrone hydrochloride 123948-87-8, Topotecan 312691-32-0, NSC 630276
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(GF inhibitors and agonists for modulating cell proliferation and cell death)

L123 ANSWER 13 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:706953 HCAPLUS
DOCUMENT NUMBER: 133:286465
TITLE: Sulfur-containing compounds and method for removal of human horny tissues
INVENTOR(S): Sun, Ying; Liu, Jue-Chen; Kimbleton, Elizabeth; Wang, Jonas C. T.
PATENT ASSIGNEE(S): Johnson and Johnson Consumer Companies, Inc., USA
SOURCE: PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000057845	A1	20001005	WO 2000-US8267	20000329 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: US 1999-126704P P 19990329 <--
US 2000-537197 A 20000329 <--

AB This invention relates to a composition and a method to facilitate phys. trimming and removing horny human tissues (e.g., a diseased nail) in a speedy and atraumatic fashion. Particularly, the invention includes a composition which softens nails comprising an effective amount of at least one sulfur-containing compound Still further, the invention contemplates a method for removing nails by applying a composition comprising an effective amount of at least one sulfur-containing compound for a duration of time sufficient to soften and removing nails by a phys. means. A kit which comprises a composition which softens nails comprises an effective amount of at least one sulfur-containing compound and at least one active agent useful in the treatment of diseased

nails. The nail swelling profiles in three compns. containing calcium thioglycolate were studied. The composition containing 5% calcium thioglycolate in water showed a lower nail swelling than the composition with 5% calcium thioglycolate and 20% urea in water. The com. **depilatory** preparation Nair lotion containing sodium thioglycolate and calcium thioglycolate behaved somewhat between the two compns., i.e., initially similar to the former, and later similar to the latter.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PI WO 2000057845 A1 **20001005**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000057845	A1	20001005	WO 2000-US8267	20000329 <--

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRAI US 1999-126704P P 19990329 <--
US 2000-537197 A 20000329 <--

AB This invention relates to a composition and a method to facilitate phys. trimming and removing horny human tissues (e.g., a diseased nail) in a speedy and atraumatic fashion. Particularly, the invention includes a composition which softens nails comprising an effective amount of at least one sulfur-containing compound Still further, the invention contemplates a method for removing nails by applying a composition comprising an effective amount of at least one sulfur-containing compound for a duration of time sufficient to soften and removing nails by a phys. means. A kit which comprises a composition which softens nails comprises an effective amount of at least one sulfur-containing compound and at least one active agent useful in the treatment of diseased nails. The nail swelling profiles in three compns. containing calcium thioglycolate were studied. The composition containing 5% calcium thioglycolate in water showed a lower nail swelling than the composition with 5% calcium thioglycolate and 20% urea in water. The com. **depilatory** preparation Nair lotion containing sodium thioglycolate and calcium thioglycolate behaved somewhat between the two compns., i.e., initially similar to the former, and later similar to the latter.

IT **Cosmetics**
(nail lacquers, antifungal; topical compns. containing sulfur compds. for removal of human horny tissues)

IT 50-23-7, Hydrocortisone 52-90-4, L-Cysteine, biological studies 53-36-1, Methylprednisolone acetate 60-23-1, Cysteamine 60-24-2, Thioethylene glycol 64-72-2, Chlortetracycline hydrochloride 64-75-5, Tetracycline hydrochloride 67-73-2, Fluocinolone acetonide 67-78-7 68-11-1, Thioglycolic acid, biological studies 70-18-8, Glutathione, biological studies 75-08-1, Thioethanol 79-42-5, Thiolactic acid 96-27-5, Thioglycerol 101-20-2, Triclocarban 108-95-2, Phenol, biological studies 121-54-0, Benzethonium chloride 136-77-6, Hexylresorcinol 147-93-3, Thiosalicylic acid 356-12-7, Fluocinonide 367-51-1, Sodium thioglycolate 382-67-2, Desoximetasone 454-29-5, Homocysteine 507-09-5, Thioacetic acid, biological studies 616-91-1, N-Acetyl-L-cysteine 814-71-1, Calcium thioglycolate 921-01-7,

D-Cysteine 1143-38-0, Anthralin 1312-73-8, Potassium sulfide 1313-82-2, Sodium sulfide, biological studies 1314-96-1, Strontium sulfide 1404-26-8, Polymyxin B 1405-10-3, Neomycin sulfate 1405-41-0, Gentamicin sulfate 1405-87-4, Bacitracin 1524-88-5, Flurandrenolide 2058-46-0, Oxytetracycline hydrochloride 2152-44-5, Betamethasone valerate 2398-96-1, Tolnaftate 2485-62-3, L-Cysteine methyl ester 3093-35-4, Halcinonide 3374-22-9, Cysteine 3380-34-5, Triclosan 3411-58-3, L-Cysteine ethyl ester 5421-46-5, Ammonium thioglycolate 5593-20-4, Betamethasone dipropionate 7553-56-2, Iodine, biological studies 7704-34-9D, Sulfur, compds., biological studies 12136-58-2, Lithium sulfide 12650-69-0, Mupirocin 13609-67-1, Hydrocortisone butyrate 20548-54-3, Calcium sulfide 22535-44-0, Lithium thioglycolate 22832-87-7, Miconazole nitrate 22916-47-8, Miconazole 23593-75-1, Clotrimazole 24583-23-1 24729-96-2, Clindamycin phosphate 25122-46-7, Clobetasol propionate 25155-18-4, Methylbenzethonium chloride 27220-47-9, Econazole 33564-31-7, Diflorasone diacetate 34452-51-2, Potassium thioglycolate 41621-49-2, Ciclopirox olamine 51022-69-6, Amcinonide 57524-89-7, Hydrocortisone valerate 60628-96-8, Bifonazole 63387-34-8, Strontium thioglycolate 63592-16-5, Magnesium thioglycolate 65277-42-1, Ketoconazole 66734-13-2 66852-54-8, Halobetasol propionate 67914-69-6, Elubiol 78613-35-1, Amorolfine 83919-23-7, Mometasone furoate 84625-61-6, Itraconazole 86386-73-4, Fluconazole 91161-71-6, Terbinafine **100986-85-4**, Levofloxacin 112965-21-6, Calcipotriene
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(topical compns. containing sulfur compds. for removal of human horny tissues)

L123 ANSWER 14 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:117139 HCAPLUS
DOCUMENT NUMBER: 132:177442
TITLE: Assembly of **telomerase** components and chaperonins and methods and compositions for **inhibiting** or stimulating **telomerase** assembly
INVENTOR(S): White, Michael A.
PATENT ASSIGNEE(S): Geron Corporation, USA
SOURCE: PCT Int. Appl., 50 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000008135	A1	20000217	WO 1999-US17724	19990805 <--
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9953381	A1	20000228	AU 1999-53381	19990805 <--

PRIORITY APPLN. INFO.: US 1998-95976P P 19980809 <--
WO 1999-US17724 W 19990805 <--

AB Methods and compns. for assembling active **telomerase** in vitro and in cells, be they in culture or in vivo , are provided, as are methods and compns. for **inhibiting** or enhancing **telomerase** activity through modulation of **telomerase** assembly. In certain preferred embodiments, methods are provided for the in vitro assembly of a **telomerase** protein component and a **telomerase** RNA component, wherein the methods involve the addition of one or more chaperonin mols., particularly substantially purified or recombinant **telomerase** chaperonins, which include the proteins hsp40, hsp70, hsp90, p23 and HOP. In such methods, one or more **telomerase** chaperonins are combined in a reaction mixture that also comprises the catalytic protein and RNA components of **telomerase.** This **invention** is based on the discovery that phosphoprotein p23 interacts and promotes assembly of **telomerase** activity, and that the hsp90 **inhibitor** geldanamycin **blocks** the enhancement of **telomerase** reconstitution. **Telomerase** activity is also enhanced by addition of heat-shock proteins 40 and 70 as well as by HOP (heat shock protein organizing protein). Screening methods for identifying **telomerase** assembly and activity **inhibitors** are also provided, along with methods for stimulating or **inhibiting telomerase** activity and assembly.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Assembly of **telomerase** components and chaperonins and methods and compositions for **inhibiting** or stimulating **telomerase** assembly

PI WO 2000008135 A1 **20000217**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2000008135	A1	20000217	WO 1999-US17724	19990805 <--

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

AU 9953381	A1	20000228	AU 1999-53381	19990805 <--
PRAI US 1998-95976P	P	19980809 <--		
WO 1999-US17724	W	19990805 <--		

AB Methods and compns. for assembling active **telomerase** in vitro and in cells, be they in culture or in vivo , are provided, as are methods and compns. for **inhibiting** or enhancing **telomerase** activity through modulation of **telomerase** assembly. In certain preferred embodiments, methods are provided for the in vitro assembly of a **telomerase** protein component and a **telomerase** RNA component, wherein the methods involve the addition of one or more chaperonin mols., particularly substantially purified or recombinant **telomerase** chaperonins, which include the proteins hsp40, hsp70, hsp90, p23 and HOP. In such methods, one or more **telomerase** chaperonins are combined in a reaction mixture that also comprises the catalytic protein and RNA components of **telomerase.** This **invention** is based on the discovery that phosphoprotein p23 interacts and promotes assembly of **telomerase** activity, and that the hsp90 **inhibitor** geldanamycin **blocks** the enhancement of **telomerase** reconstitution. **Telomerase**

activity is also enhanced by addition of heat-shock proteins 40 and 70 as well as by HOP (heat shock protein organizing protein). Screening methods for identifying **telomerase** assembly and activity **inhibitors** are also provided, along with methods for stimulating or **inhibiting telomerase** activity and assembly.

IT Proteins, specific or class
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(HOP (heat-shock protein-organizing protein); assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Heat-shock proteins
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(HSP 70; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Heat-shock proteins
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(HSP 90; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Animal cell line
(HT-1080; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Nervous system
(Huntington's chorea, treatment of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Animal cell
Anti-Alzheimer's agents
Anti-infective agents
Antiparkinsonian agents
Antitumor agents
Bird (Aves)
Cat (Felis catus)
Cattle
Dog (Canis familiaris)
Drug screening
Drugs
Gene therapy
Horse (Equus caballus)
Molecular association
Sheep
Swine
Vertebrate (Vertebrata)
(assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Antisense oligonucleotides
Ribozymes
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Joint, anatomical
(degeneration, treatment of; assembly of **telomerase** components and chaperonins and methods and compns. for

inhibiting or stimulating **telomerase** assembly)

IT Blood vessel
(endothelium, treatment of conditions associated with replicative capacity of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT **Hair**
(**follicle**, treatment of conditions associated with replicative capacity of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Heat-shock proteins
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(hsp 40; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Antitumor agents
(leukemia; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Eye, disease
(macula, degeneration, treatment of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Cell proliferation
(modulating disorders of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Hematopoietic precursor cell
Lymphocyte
(natural killer cell, treatment of conditions associated with replicative capacity of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Bone marrow
(osteoprogenitor cell, treatment of conditions associated with replicative capacity of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Eye
(pigment epithelium, treatment of conditions associated with replicative capacity of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Brain, disease
(stroke, treatment of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT Chaperonins
RNA
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(**telomerase** component; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT B cell (lymphocyte)
Basophil
Chondrocyte
Fibroblast

Monocyte
Neutrophil
Osteoblast
T cell (lymphocyte)
(treatment of conditions associated with replicative capacity of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT **Alopecia**
Cell aging
(treatment of; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT 30562-34-6, Geldanamycin
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT **120178-12-3, Telomerase** reverse transcriptase
RL: BPR (Biological process); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)
(assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

IT 197183-99-6 243940-92-3, 4: PN: WO0008135 SEQID: 6 unclaimed DNA 243940-93-4, 3: PN: WO0008135 SEQID: 5 unclaimed DNA 259238-19-2, 2: PN: WO0008135 SEQID: 4 unclaimed DNA
RL: PRP (Properties)
(unclaimed nucleotide sequence; assembly of **telomerase** components and chaperonins and methods and compns. for **inhibiting** or stimulating **telomerase** assembly)

L123 ANSWER 15 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 2000:83232 HCAPLUS
DOCUMENT NUMBER: 132:127477
TITLE: Cosmetic and dermatological preparations with an effective content of bile acids, their salts or derivatives
INVENTOR(S): Schreiner, Volker; Lanzendoerfer, Ghita
PATENT ASSIGNEE(S): Beiersdorf A.-G., Germany
SOURCE: Ger. Offen., 12 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 19834814	A1	20000203	DE 1998-19834814	19980801 <--
WO 2000007557	A1	20000217	WO 1999-EP5157	19990720 <--
W: JP, US				
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
EP 1100455	A1	20010523	EP 1999-938295	19990720 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2003526602	T2	20030909	JP 2000-563243	19990720 <--

PRIORITY APPLN. INFO.: DE 1998-19834814 A 19980801 <--

WO 1999-EP5157 W 19990720 <--

AB Topical application of prepns. containing bile acids, their salts and/or derivs. restores or reinforces the barrier function of the skin, counteracts skin drying and aging, and protects the skin from environmental influences. Thus, a gel contained sucrose stearate 3.00, cetearyl alc. 2.00, deoxycholic acid 0.02, Carbopol 0.50, glycerin 3.00, antioxidants, preservatives, neutralizing agents, perfume, dyes, and H2O to 100 weight%.

PI DE 19834814 A1 **20000203**

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	DE 19834814	A1	20000203	DE 1998-19834814	19980801	<--
	WO 2000007557	A1	20000217	WO 1999-EP5157	19990720	<--
	W: JP, US					
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
	EP 1100455	A1	20010523	EP 1999-938295	19990720	<--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
	JP 2003526602	T2	20030909	JP 2000-563243	19990720	<--
PRAI	DE 1998-19834814	A	19980801	<--		
	WO 1999-EP5157	W	19990720	<--		

IT **Cosmetics**
(barrier creams; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Cosmetics**
(barrier gels; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Cosmetics**
Hair preparations
(**conditioners**; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Shampoos**
(conditioning; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Cosmetics**
Drug delivery systems
(emulsions; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Cosmetics**
(lipsticks; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Cosmetics**
Drug delivery systems
(lotions; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Cosmetics**
(makeups; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Bath preparations**
(oils; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Cosmetics**
Drug delivery systems
(oily; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Antiperspirants**
(roll-on; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT **Cosmetics**

Drug delivery systems
(sprays; cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

IT 81-23-2, Dehydrocholic acid 81-24-3, Taurocholic acid 83-44-3, Deoxycholic acid **128-13-2**, Ursodeoxycholic acid 434-13-9, Lithocholic acid 475-31-0, Glycocholic acid 516-50-7, Taurodeoxycholic acid 516-90-5, Taurolithocholic acid
RL: BUU (Biological use, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(cosmetic and dermatol. prepns. containing bile acids, their salts or derivs.)

L123 ANSWER 16 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:390368 HCAPLUS
DOCUMENT NUMBER: 131:39760
TITLE: Methods for treatment of neuro- and nephro-disorders and therapeutic toxicities using amifostine and other aminothiol compounds
INVENTOR(S): Stogniew, Martin; Alberts, David S.; Kaplan, Edward H.
PATENT ASSIGNEE(S): U.S. Bioscience, Inc., USA; The Arizona Board of Regents
SOURCE: PCT Int. Appl., 48 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9929312	A1	19990617	WO 1998-US26096	19981209 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 5994409	A	19991130	US 1997-987550	19971209 <--
CA 2313089	AA	19990617	CA 1998-2313089	19981209 <--
AU 9917184	A1	19990628	AU 1999-17184	19981209 <--
AU 739068	B2	20011004		
EP 1039887	A1	20001004	EP 1998-962011	19981209 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 2001525359	T2	20011211	JP 2000-523983	19981209 <--
BR 9813524	A	20020219	BR 1998-13524	19981209 <--

PRIORITY APPLN. INFO.: US 1997-987550 A 19971209 <--
WO 1998-US26096 W 19981209 <--

OTHER SOURCE(S): MARPAT 131:39760

AB S-2-(3-aminopropylamino)ethyl dihydrogen phosphorothioate (amifostine) and other aminothiol compds. are used to treat and reverse toxicities caused by therapeutic agents, radiation treatment or diabetes. A method is provided for treating neurotoxicity and nephrotoxicity associated with the administration of chemotherapeutic agents.

REFERENCE COUNT: 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PI WO 9929312 A1 **19990617**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE

PI	WO 9929312	A1	19990617	WO 1998-US26096	19981209 <--
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	US 5994409	A	19991130	US 1997-987550	19971209 <--
	CA 2313089	AA	19990617	CA 1998-2313089	19981209 <--
	AU 9917184	A1	19990628	AU 1999-17184	19981209 <--
	AU 739068	B2	20011004		
	EP 1039887	A1	20001004	EP 1998-962011	19981209 <--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2001525359	T2	20011211	JP 2000-523983	19981209 <--
	BR 9813524	A	20020219	BR 1998-13524	19981209 <--
PRAI	US 1997-987550	A	19971209 <--		
	WO 1998-US26096	W	19981209 <--		

IT **Alopecia**
Anti-AIDS agents
Antibiotics
Antidiabetic agents
Antihypertensives
Antitumor agents
Antiviral agents
Diabetes mellitus
Fungicides
Kidney, disease
Neoplasm
Nervous system agents
Radioprotectants
Radiotherapy
X-ray
(amifostine and other aminothiols for treatment of neuro- and nephro-disorders and therapeutic toxicities)

IT 57-22-7, Vincristine 865-21-4, Vinblastine 1397-89-3, Amphotericin B 1403-66-3, Gentamicin 1404-90-6, Vancomycin **3056-17-5**, d4T 7481-89-2, DdC 8063-07-8, Kanamycin 15663-27-1, Cisplatin 20830-81-3, Daunorubicin 23214-92-8, Doxorubicin 30516-87-1, AZT 32986-56-4, Tobramicin 33069-62-4, Paclitaxel 33419-42-0, Etoposide 37517-28-5, Amikacin 41575-94-4, Carboplatin **69655-05-6**, DdI 95058-81-4, Gemcitabine 114977-28-5, Docetaxel 125317-39-7, Navelbine 134678-17-4, 3TC
RL: ADV (Adverse effect, including toxicity); BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amifostine and other aminothiols for treatment of neuro- and nephro-disorders and therapeutic toxicities)

L123 ANSWER 17 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1999:204212 HCAPLUS
DOCUMENT NUMBER: 130:336210
TITLE: Longevity, stress response, and cancer in aging telomerase-deficient mice
AUTHOR(S): Rudolph, Karl Lenhard; Chang, Sandy; Lee, Han-Woong; Blasco, Maria; Gottlieb, Geoffrey J.; Greider, Carol; DePinho, Ronald A.

CORPORATE SOURCE: Department of Adult Oncology, Dana Farber Cancer Institute, Boston, MA, 02115, USA
SOURCE: Cell (Cambridge, Massachusetts) (**1999**), 96(5), 701-712
CODEN: CELLB5; ISSN: 0092-8674
PUBLISHER: Cell Press
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Telomere maintenance is thought to play a role in signaling cellular senescence; however, a link with organismal aging processes has not been established. The **telomerase** null mouse provides an opportunity to understand the effects associated with critical telomere shortening at the organismal level. We studied a variety of physiol. processes in an aging cohort of mTR-/- mice. Loss of telomere function did not elicit a full spectrum of classical pathophysiol. symptoms of aging. However, age-dependent telomere shortening and accompanying genetic instability were associated with shortened life span as well as a **reduced** capacity to respond to stresses such as wound healing and hematopoietic ablation. In addition, we found an increased incidence of spontaneous malignancies. These findings demonstrate a critical role for telomere length in the overall fitness, reserve, and well being of the aging organism.

REFERENCE COUNT: 66 THERE ARE 66 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

SO Cell (Cambridge, Massachusetts) (**1999**), 96(5), 701-712
CODEN: CELLB5; ISSN: 0092-8674

AB Telomere maintenance is thought to play a role in signaling cellular senescence; however, a link with organismal aging processes has not been established. The **telomerase** null mouse provides an opportunity to understand the effects associated with critical telomere shortening at the organismal level. We studied a variety of physiol. processes in an aging cohort of mTR-/- mice. Loss of telomere function did not elicit a full spectrum of classical pathophysiol. symptoms of aging. However, age-dependent telomere shortening and accompanying genetic instability were associated with shortened life span as well as a **reduced** capacity to respond to stresses such as wound healing and hematopoietic ablation. In addition, we found an increased incidence of spontaneous malignancies. These findings demonstrate a critical role for telomere length in the overall fitness, reserve, and well being of the aging organism.

IT **Hair**
(graying; telomerase null mouse model to understand pathol. effects associated with critical telomere shortening)

IT Aging, animal
Alopecia
Cataract
Longevity
Lymphoma
Sarcoma
Stress, animal
Telomeres (chromosome)
Transformation, neoplastic
Wound healing
(telomerase null mouse model to understand pathol. effects associated with critical telomere shortening)

L123 ANSWER 18 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:621122 HCAPLUS
DOCUMENT NUMBER: 129:239917
TITLE: Oxyalkylene phosphate compounds and therapeutic uses thereof
INVENTOR(S): Nudelman, Abraham; Rephaeli, Ada

PATENT ASSIGNEE(S): Beacon Laboratories, L.L.C., USA
SOURCE: PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 9840080	A1	19980917	WO 1998-US4834	19980311	<--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
US 6030961	A	20000229	US 1997-814386	19970311	<--
AU 9864597	A1	19980929	AU 1998-64597	19980311	<--
EP 986391	A1	20000322	EP 1998-910333	19980311	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					
JP 2001514665	T2	20010911	JP 1998-539793	19980311	<--

PRIORITY APPLN. INFO.: US 1997-814386 A 19970311 <--
WO 1998-US4834 W 19980311 <--

OTHER SOURCE(S): MARPAT 129:239917

AB Compns. and methods are provided for treating, **preventing** or ameliorating cancer and other proliferative diseases, as are methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and in particular, augmenting expression of tumor suppressor genes, inducing tolerance to antigens, treating, **preventing** or ameliorating protozoan infection, or **inhibiting** histone deacetylase in cells. The compns. of the **invention** are to and the methods of the **invention** use oxyalkalene phosphate compds.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PI WO 9840080 A1 **19980917**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI WO 9840080	A1	19980917	WO 1998-US4834	19980311	<--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
US 6030961	A	20000229	US 1997-814386	19970311	<--
AU 9864597	A1	19980929	AU 1998-64597	19980311	<--
EP 986391	A1	20000322	EP 1998-910333	19980311	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					

JP 2001514665 T2 20010911 JP 1998-539793 19980311 <--
PRAI US 1997-814386 A 19970311 <--
WO 1998-US4834 W 19980311 <--
AB Compns. and methods are provided for treating, **preventing** or ameliorating cancer and other proliferative diseases, as are methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and in particular, augmenting expression of tumor suppressor genes, inducing tolerance to antigens, treating, **preventing** or ameliorating protozoan infection, or **inhibiting** histone deacetylase in cells. The compns. of the **invention** are to and the methods of the **invention** use oxyalkalene phosphate compds.
ST oxyalkylene phosphate prepn therapeutic; antitumor antiproliferative wound healing oxyalkylene phosphate; cutaneous ulcer gastrointestinal disorder oxyalkylene phosphate; blood disorder gene expression oxyalkylene phosphate; immunomodulation antidiabetic cystic fibrosis oxyalkylene phosphate; **telomerase inhibition** antigen tolerance oxyalkylene phosphate; antiprotozoal histone deacetylase **inhibition** oxyalkylene phosphate
IT **Hair**
(**follicle**, epithelial cells; oxyalkylene phosphate compds. and therapeutic use)

L123 ANSWER 19 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:621109 HCAPLUS
DOCUMENT NUMBER: 129:239915
TITLE: Metabolically stabilized oxyalkylene esters and therapeutic uses thereof
INVENTOR(S): Nudelman, Abraham; Rephaeli, Ada; Neiss, Edward; Loev, Bernard
PATENT ASSIGNEE(S): Beacon Laboratories L.L.C., USA
SOURCE: PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9840066	A1	19980917	WO 1998-US4753	19980311 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6110955	A	20000829	US 1997-814975	19970311 <--
AU 9864579	A1	19980929	AU 1998-64579	19980311 <--
EP 986380	A1	20000322	EP 1998-910307	19980311 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				

PRIORITY APPLN. INFO.: US 1997-814975 A 19970311 <--
WO 1998-US4753 W 19980311 <--

OTHER SOURCE(S): MARPAT 129:239915

AB Compns. for and methods of treating, **preventing** or ameliorating cancer and other proliferative diseases are disclosed, as are methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and particularly augmenting expression of a tumor suppressor gene, inducing tolerance to an antigen and treating, ameliorating or **preventing** protozoan infection. The methods of the **invention** use metabolically stabilized oxyalkylene esters.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PI WO 9840066 A1 **19980917**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI WO 9840066	A1	19980917	WO 1998-US4753	19980311	<--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
US 6110955	A	20000829	US 1997-814975	19970311	<--
AU 9864579	A1	19980929	AU 1998-64579	19980311	<--
EP 986380	A1	20000322	EP 1998-910307	19980311	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					

PRAI US 1997-814975 A 19970311 <--
WO 1998-US4753 W 19980311 <--

AB Compns. for and methods of treating, **preventing** or ameliorating cancer and other proliferative diseases are disclosed, as are methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and particularly augmenting expression of a tumor suppressor gene, inducing tolerance to an antigen and treating, ameliorating or **preventing** protozoan infection. The methods of the **invention** use metabolically stabilized oxyalkylene esters.

ST metabolically stabilized oxyalkylene ester therapeutic; antitumor antiproliferative oxyalkylene ester; gastrointestinal blood disorder anemia oxyalkylene ester; immunomodulation gene expression diabetes oxyalkylene ester; cystic fibrosis **telomerase inhibition** oxyalkylene ester; virus assocd tumor oxyalkylene ester; antigen tolerance protozoan antiinfective oxyalkylene ester; suppressor tumor gene expression oxyalkylene ester

IT **Hair**
(**follicle**, epithelial cells; metabolically stabilized oxyalkylene esters and therapeutic uses thereof)

IT 9076-57-7, Histone deacetylase **120178-12-3**, **Telomerase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**inhibitors**; metabolically stabilized oxyalkylene esters and therapeutic uses thereof)

L123 ANSWER 20 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:621108 HCAPLUS
DOCUMENT NUMBER: 129:239914
TITLE: Hydroxy- and ether-containing oxyalkylene esters and therapeutic uses thereof
INVENTOR(S): Nudelman, Abraham; Rephaeli, Adi
PATENT ASSIGNEE(S): Beacon Laboratories, L.L.C., USA
SOURCE: PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9840065	A1	19980917	WO 1998-US4764	19980311 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
US 6043389	A	20000328	US 1997-814224	19970311 <--
AU 9865501	A1	19980929	AU 1998-65501	19980311 <--
AU 728419	B2	20010111		
EP 998278	A1	20000510	EP 1998-911574	19980311 <--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
JP 2001514664	T2	20010911	JP 1998-539760	19980311 <--
US 6239176	B1	20010529	US 2000-504786	20000215 <--

PRIORITY APPLN. INFO.: US 1997-814224 A 19970311 <--
WO 1998-US4764 W 19980311 <--
OTHER SOURCE(S): MARPAT 129:239914

AB This **invention** relates to compns. for and methods of treating, **preventing** or ameliorating cancer and other proliferative diseases as well as methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, augmenting expression of tumor suppressor genes, inducing tolerance to antigens, or treating, **preventing** or ameliorating protozoan infection or **inhibiting** histone deacetylase in cells. The compns. of the **invention** are to and the methods of the **invention** use hydroxy and ether-containing oxyalkylene esters.

REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PI WO 9840065 A1 **19980917**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9840065	A1	19980917	WO 1998-US4764	19980311 <--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO,				

NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

US 6043389	A	20000328	US 1997-814224	19970311	<--
AU 9865501	A1	19980929	AU 1998-65501	19980311	<--
AU 728419	B2	20010111			
EP 998278	A1	20000510	EP 1998-911574	19980311	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					
JP 2001514664	T2	20010911	JP 1998-539760	19980311	<--
US 6239176	B1	20010529	US 2000-504786	20000215	<--
PRAI US 1997-814224	A	19970311	<--		
WO 1998-US4764	W	19980311	<--		

AB This **invention** relates to compns. for and methods of treating, **preventing** or ameliorating cancer and other proliferative diseases as well as methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, augmenting expression of tumor suppressor genes, inducing tolerance to antigens, or treating, **preventing** or ameliorating protozoan infection or **inhibiting** histone deacetylase in cells. The compns. of the **invention** are to and the methods of the **invention** use hydroxy and ether-containing oxyalkylene esters.

ST hydroxy ether oxyalkylene ester prepn therapeutic; antitumor antiproliferative wound healing oxyalkylene ester; cutaneous ulcer GI disorder oxyalkylene ester; blood disorder anemia immunomodulation oxyalkylene ester; gene expression diabetes oxyalkylene ester; cystic fibrosis **telomerase inhibition** oxyalkylene ester; immune tolerance protozoan antiinfective oxyalkylene ester; histone deacetylase **inhibition** oxyalkylene ester; tumor suppressor gene expression oxyalkylene ester

IT **Hair**
(**follicle**, epithelial cells; hydroxy- and ether-containing oxyalkylene esters and therapeutic uses thereof)

L123 ANSWER 21 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:621086 HCAPLUS
DOCUMENT NUMBER: 129:239911
TITLE: Nitrogen-containing oxyalkylene esters and therapeutic uses thereof
INVENTOR(S): Nudelman, Abraham; Rephaeli, Ada
PATENT ASSIGNEE(S): Beacon Laboratories, L.L.C., USA
SOURCE: PCT Int. Appl., 96 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 9839966	A1	19980917	WO 1998-US4763	19980311	<--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP,					

KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO,
NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA,
UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI,
FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM,
GA, GN, ML, MR, NE, SN, TD, TG

US 6110970	A	20000829	US 1997-814225	19970311	<--
AU 9865500	A1	19980929	AU 1998-65500	19980311	<--
EP 973389	A1	20000126	EP 1998-911573	19980311	<--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO

PRIORITY APPLN. INFO.: US 1997-814225 A 19970311 <--
WO 1998-US4763 W 19980311 <--

OTHER SOURCE(S): MARPAT 129:239911

AB Compns. and methods are provided for treating, **preventing** or ameliorating cancer and other proliferative diseases, as are methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and particularly augmenting expression of tumor suppressor genes, inducing tolerance to antigens, treating, **preventing** or ameliorating protozoan infection or **inhibiting** histone deacetylase in cells. The compns. of the **invention** are to and the methods of the **invention** use nitrogen-containing oxyalkyl esters.

REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PI WO 9839966 A1 **19980917**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI WO 9839966	A1	19980917	WO 1998-US4763	19980311	<--

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP,
KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO,
NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA,
UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI,
FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM,
GA, GN, ML, MR, NE, SN, TD, TG

US 6110970	A	20000829	US 1997-814225	19970311	<--
AU 9865500	A1	19980929	AU 1998-65500	19980311	<--
EP 973389	A1	20000126	EP 1998-911573	19980311	<--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
IE, SI, LT, LV, FI, RO

PRAI US 1997-814225	A	19970311	<--
WO 1998-US4763	W	19980311	<--

AB Compns. and methods are provided for treating, **preventing** or ameliorating cancer and other proliferative diseases, as are methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and particularly augmenting expression of tumor suppressor genes, inducing tolerance to antigens, treating, **preventing** or ameliorating protozoan infection or **inhibiting** histone deacetylase in cells. The compns. of the **invention** are to and the methods of the

invention use nitrogen-containing oxyalkyl esters.

ST nitrogen contg oxyalkyl ester prepn therapeutic; antitumor antiproliferative nitrogen contg oxyalkyl ester; wound healing nitrogen contg oxyalkyl ester; cutaneous ulcer nitrogen contg oxyalkyl ester; gastrointestinal disorder nitrogen contg oxyalkyl ester; blood disorder nitrogen contg oxyalkyl ester; immunomodulation antidiabetic nitrogen contg oxyalkyl ester; gene expression nitrogen contg oxyalkyl ester; cystic fibrosis nitrogen contg oxyalkyl ester; **telomerase inhibition** nitrogen contg oxyalkyl ester; antigen tolerance nitrogen contg oxyalkyl ester; antiprotozoal nitrogen contg oxyalkyl ester; histone deacetylase **inhibition** oxyalkyl ester

IT **Hair**
(**follicle**, epithelial cells; nitrogen-containing oxyalkylene esters and therapeutic use)

L123 ANSWER 22 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:621085 HCAPLUS
DOCUMENT NUMBER: 129:255005
TITLE: Unsaturated oxyalkylene esters and therapeutic uses thereof
INVENTOR(S): Neiss, Edward; Loev, Bernard
PATENT ASSIGNEE(S): Beacon Laboratories L.L.C., USA
SOURCE: PCT Int. Appl., 57 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 9839965	A1	19980917	WO 1998-US4756	19980311	<--
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
US 6124495	A	20000926	US 1997-814366	19970311	<--
AU 9865496	A1	19980929	AU 1998-65496	19980311	<--
AU 746268	B2	20020418			
EP 973388	A1	20000126	EP 1998-911569	19980311	<--
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					
US 6599937	B1	20030729	US 2000-669013	20000925	<--

PRIORITY APPLN. INFO.: US 1997-814366 A 19970311 <--
WO 1998-US4756 W 19980311 <--

OTHER SOURCE(S): MARPAT 129:255005

AB Compns. and methods are provided for treating, **preventing**, or ameliorating cancer and other proliferative diseases, are methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and particularly augmenting expression of a tumor suppressor gene and inducing tolerance to an antigen. The methods of the **invention** use

unsatd. oxyalkylene esters.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PI WO 9839965 A1 **19980917**

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI	WO 9839965	A1	19980917	WO 1998-US4756	19980311	<--
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM					
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG					
	US 6124495	A	20000926	US 1997-814366	19970311	<--
	AU 9865496	A1	19980929	AU 1998-65496	19980311	<--
	AU 746268	B2	20020418			
	EP 973388	A1	20000126	EP 1998-911569	19980311	<--
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO					
	US 6599937	B1	20030729	US 2000-669013	20000925	<--
PRAI	US 1997-814366	A	19970311	<--		
	WO 1998-US4756	W	19980311	<--		

AB Compns. and methods are provided for treating, **preventing**, or ameliorating cancer and other proliferative diseases, are methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and particularly augmenting expression of a tumor suppressor gene and inducing tolerance to an antigen. The methods of the **invention** use unsatd. oxyalkylene esters.

ST unsatd oxyalkylene ester prepn therapeutic; antitumor antiproliferative unsatd oxyalkylene ester; wound healing unsatd oxyalkylene ester; cutaneous ulcer unsatd oxyalkylene ester; blood disorder immunomodulation unsatd oxyalkylene ester; gene expression diabetes unsatd oxyalkylene ester; cystic fibrosis unsatd oxyalkylene ester; **telomerase inhibition** unsatd oxyalkylene ester; immune tolerance unsatd oxyalkylene ester

IT **Hair**
(**follicle**, epithelial cells; unsatd. oxyalkylene esters and therapeutic use)

L123 ANSWER 23 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1998:484940 HCAPLUS
DOCUMENT NUMBER: 129:104235
TITLE: Tricarboxylic acid-containing oxyalkyl esters, and therapeutic uses thereof
INVENTOR(S): Nudelman, Abraham; Rephaeli, Ada
PATENT ASSIGNEE(S): Beacon Laboratories L.L.C., USA
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
WO 9829114	A1	19980709	WO 1997-US23725	19971230	<--

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

US 6130248	A	20001010	US 1996-781905	19961230	<--
AU 9856173	A1	19980731	AU 1998-56173	19971230	<--
EP 961614	A1	19991208	EP 1997-952599	19971230	<--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

PRIORITY APPLN. INFO.:			US 1996-781905	A 19961230	<--
			US 1997-814365	A 19970311	<--
			WO 1997-US23725	W 19971230	<--

OTHER SOURCE(S): MARPAT 129:104235

AB Compns. for and methods of treating, **preventing** or ameliorating cancer and other proliferative diseases are provided, as are methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and particularly augmenting expression of tumor suppressor genes, inducing tolerance to antigens; treating, **preventing**, or ameliorating protozoan infection or **inhibiting** histone deacetylase in cells. The methods of the **invention** use tricarboxylic acid substituted oxyalkyl esters.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

PI WO 9829114 A1 **19980709**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI WO 9829114	A1	19980709	WO 1997-US23725	19971230	<--

W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

US 6130248	A	20001010	US 1996-781905	19961230	<--
AU 9856173	A1	19980731	AU 1998-56173	19971230	<--
EP 961614	A1	19991208	EP 1997-952599	19971230	<--

R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO

PRAI US 1996-781905	A	19961230	<--
US 1997-814365	A	19970311	<--
WO 1997-US23725	W	19971230	<--

AB Compns. for and methods of treating, **preventing** or ameliorating cancer and other proliferative diseases are provided, as are methods of inducing wound healing, treating cutaneous ulcers, treating gastrointestinal disorders, treating blood disorders such as anemias, immunomodulation, enhancing recombinant gene expression, treating

insulin-dependent patients, treating cystic fibrosis patients, **inhibiting telomerase** activity, treating virus-associated tumors, especially EBV-associated tumors, modulating gene expression and particularly augmenting expression of tumor suppressor genes, inducing tolerance to antigens; treating, **preventing**, or ameliorating protozoan infection or **inhibiting** histone deacetylase in cells. The methods of the **invention** use tricarboxylic acid substituted oxyalkyl esters.

IT **Hair**
(**follicle**; tricarboxylic acid-containing oxyalkyl esters, and therapeutic uses thereof)

L123 ANSWER 24 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1996:458190 HCAPLUS
DOCUMENT NUMBER: 125:164258
TITLE: Disruption of cholesterol 7α-hydroxylase gene in mice. II. Bile acid deficiency is overcome by induction of oxysterol 7α-hydroxylase
AUTHOR(S): Schwarz, Margrit; Lund, Erik G.; Setchell, Kenneth D. R.; Kayden, Herbert J.; Zerwekh, Joseph E.; Bjorkhem, Ingemar; Herz, Joachim; Russell, David W.
CORPORATE SOURCE: Dep. Mol. Genetics Int. Med., Univ. Texas Southwestern Med. Cent., Dallas, TX, 75235-9046, USA
SOURCE: Journal of Biological Chemistry (**1996**), 271(30), 18024-18031
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Past expts. and current paradigms of cholesterol homeostasis suggest that cholesterol 7α-hydroxylase plays a crucial role in sterol metabolism by controlling the conversion of cholesterol into bile acids. Consistent with this conclusion, we show in the accompanying paper that mice deficient in cholesterol 7α-hydroxylase (Cyp7-/- mice) exhibit a complex phenotype consisting of abnormal lipid excretion, skin pathologies, and behavioral irregularities. Aspects of lipid metabolism in the Cyp7-/- mice are characterized here to deduce the physiol. basis of this phenotype. Serum lipid, cholesterol, and lipoprotein contents are indistinguishable between wild-type and Cyp7-/- mice. Vitamin D3 and E levels are low to undetectable in knockout animals. Stool fat content is significantly elevated in newborn Cyp7-/- mice and gradually declines to wild-type levels at 28 days of age. Several species of 7α-hydroxylated bile acids are detected in the bile and stool of adult Cyp7-/- animals. A hepatic oxysterol 7α-hydroxylase enzyme activity that may account for the 7α-hydroxylated bile acids is induced between days 21 and 30 in both wild-type and deficient mice. An anomalous oily coat in the Cyp7-/- animals is due to the presence of excess monoglyceride esters in the fur. These data show that 7α-hydroxylase and the pathway of bile acid synthesis initiated by this enzyme are essential for proper absorption of dietary lipids and fat-soluble vitamins in newborn mice, but not for the maintenance of serum cholesterol and lipid levels. In older animals, an alternate pathway of bile acid synthesis involving an inducible oxysterol 7α-hydroxylase plays a crucial role in lipid and bile acid metabolism

SO Journal of Biological Chemistry (**1996**), 271(30), 18024-18031
CODEN: JBCHA3; ISSN: 0021-9258

IT **Hair**
(oily, oxysterol 7α-hydroxylase role in lipid and bile acid metabolism in relation to senescence)

IT 67-97-0, Vitamin D3 81-25-4 83-44-3 83-49-8 **128-13-2**
434-13-9 474-25-9 1406-18-4, Vitamin E 2393-58-0 2393-59-1
2464-18-8 2569-04-2 5130-29-0 6830-03-1 7170-94-7 63324-19-6
87638-62-8
RL: BOC (Biological occurrence); BSU (Biological study, unclassified);
BIOL (Biological study); OCCU (Occurrence)
(oxysterol 7α-hydroxylase role in lipid and bile acid metabolism in relation to senescence)

L123 ANSWER 25 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1995:423934 HCAPLUS
DOCUMENT NUMBER: 122:177620
TITLE: Time course of appearance of ofloxacin in human scalp **hair** after oral administration
AUTHOR(S): Uematsu, Toshihiko; Kosuge, Kazuhiro; Araki, Sei-ichi; Ishiye, Masayuki; Asai, Yoshihiro; Nakashima, Mitsuyoshi
CORPORATE SOURCE: School of Medicine, Hamamatsu University, Hamamatsu, Japan
SOURCE: Therapeutic Drug Monitoring (**1995**), 17(1), 101-3
CODEN: TDMODV; ISSN: 0163-4356
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The time course of appearance of antimicrobial ofloxacin (OFLX) in human scalp **hair** was monitored in three healthy male volunteers after the oral administration of 100 mg OFLX three times daily for 2 consecutive days. **Hair** samples were collected from each subject by plucking several strands of frontal **hair** every day from 1 till 16 days after administration. A single **hair** was dissolved in 1 M NaOH to extract OFLX by chloroform, and the drug was measured by high-performance liquid chromatog. and fluorescence detection. OFLX started to appear in the **hair** 1 to 3 days after administration and reached the maximal level approx. 4 to 9 days, remaining at almost the same level thereafter. This finding suggests the **slow** transfer of OFLX from **hair follicle** cells to **hair** matrix may be due to the slow dissociation of OFLX from bound melanin.
TI Time course of appearance of ofloxacin in human scalp **hair** after oral administration
SO Therapeutic Drug Monitoring (**1995**), 17(1), 101-3
CODEN: TDMODV; ISSN: 0163-4356
AB The time course of appearance of antimicrobial ofloxacin (OFLX) in human scalp **hair** was monitored in three healthy male volunteers after the oral administration of 100 mg OFLX three times daily for 2 consecutive days. **Hair** samples were collected from each subject by plucking several strands of frontal **hair** every day from 1 till 16 days after administration. A single **hair** was dissolved in 1 M NaOH to extract OFLX by chloroform, and the drug was measured by high-performance liquid chromatog. and fluorescence detection. OFLX started to appear in the **hair** 1 to 3 days after administration and reached the maximal level approx. 4 to 9 days, remaining at almost the same level thereafter. This finding suggests the **slow** transfer of OFLX from **hair follicle** cells to **hair** matrix may be due to the slow dissociation of OFLX from bound melanin.
ST ofloxacin pharmacokinetics **hair**
IT **Hair**
Legal chemistry and medicine
(time course of appearance of ofloxacin in human scalp **hair** after oral administration)
IT **82419-36-1**, Ofloxacin

RL: BOC (Biological occurrence); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence)
(time course of appearance of ofloxacin in human scalp **hair** after oral administration)

L123 ANSWER 26 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1995:267825 HCAPLUS
DOCUMENT NUMBER: 122:45520
TITLE: Using ofloxacin as a time marker in **hair** analysis for monitoring the dosage history of haloperidol
AUTHOR(S): Nakano, M.; Uematsu, T.; Sato, H.; Kosuge, K.; Nishimoto, M.; Nakashima, M.
CORPORATE SOURCE: School of Medicine, Hamamatsu University, Hamamatsu, 431-31, Japan
SOURCE: European Journal of Clinical Pharmacology (**1994**), 47(2), 195-202
CODEN: EJCPAS; ISSN: 0031-6970
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Hair** samples were obtained 1-5 mo after ingestion of the antimicrobial ofloxacin, which had been given for 1 or 3 days at the commencement of haloperidol administration, or when its dosage was reduced. The axial distribution of ofloxacin, haloperidol and its active metabolite, reduced haloperidol, was analyzed in segments from single strands of **hair.** Ofloxacin was detected where the content of haloperidol and **reduced** haloperidol along the **hair** shaft showed a sharp change, corresponding to the change in dose. When we matched the time scale of the dosage history to the growth rate, which was estimated using ofloxacin as the time marker, the distribution of the haloperidol and reduced haloperidol precisely coincided with the rise and fall in the dose of haloperidol. These findings demonstrate that ofloxacin can serve as a time marker when drug distribution along the **hair** shaft is used to obtain the drug exposure history of an individual.

TI Using ofloxacin as a time marker in **hair** analysis for monitoring the dosage history of haloperidol
SO European Journal of Clinical Pharmacology (**1994**), 47(2), 195-202
CODEN: EJCPAS; ISSN: 0031-6970

AB **Hair** samples were obtained 1-5 mo after ingestion of the antimicrobial ofloxacin, which had been given for 1 or 3 days at the commencement of haloperidol administration, or when its dosage was reduced. The axial distribution of ofloxacin, haloperidol and its active metabolite, reduced haloperidol, was analyzed in segments from single strands of **hair.** Ofloxacin was detected where the content of haloperidol and **reduced** haloperidol along the **hair** shaft showed a sharp change, corresponding to the change in dose. When we matched the time scale of the dosage history to the growth rate, which was estimated using ofloxacin as the time marker, the distribution of the haloperidol and reduced haloperidol precisely coincided with the rise and fall in the dose of haloperidol. These findings demonstrate that ofloxacin can serve as a time marker when drug distribution along the **hair** shaft is used to obtain the drug exposure history of an individual.

ST haloperidol **hair** analysis ofloxacin time marker; forensic analysis haloperidol **hair** ofloxacin
IT **Hair**
Legal chemistry and medicine
(ofloxacin as a time marker in **hair** anal. for haloperidol dosage history monitoring)

IT 52-86-8, Haloperidol **82419-36-1**, Ofloxacin
RL: ANT (Analyte); ANST (Analytical study)
(ofloxacin as a time marker in **hair** anal. for haloperidol dosage history monitoring)

L123 ANSWER 27 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1994:235277 HCAPLUS
DOCUMENT NUMBER: 120:235277
TITLE: Simultaneous determination of ofloxacin, norfloxacin and ciprofloxacin in human **hair** by high-performance liquid chromatography and fluorescence detection
AUTHOR(S): Mizuno, Atsuhiro; Uematsu, Toshihiko; Nakashima, Mitsuyoshi
CORPORATE SOURCE: Sch. Med., Uamamatsu Univ., Hamamatsu, 431-31, Japan
SOURCE: Journal of Chromatography, B: Biomedical Sciences and Applications (**1994**), 653(2), 187-93
CODEN: JCBBEP; ISSN: 1387-2273
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A high-performance liquid chromatog. method for the simultaneous determination of ofloxacin, norfloxacin and ciprofloxacin in human **hair** is described. A reversed-phase C18 column and a fluorescence detector with switching fluorescence wavelengths were used together with solid-phase extraction of the drugs from **hair** dissolved in 1 M sodium hydroxide. Reproducibility and linearity studies yielded coeffs. of variations of 0.2-2.2, 1.4-3.1 and 1.5-3.4%, and correlation coeffs. of 1.000, 0.999 and 0.999 within the concentration range 0.3-100 ng/mL for ofloxacin, norfloxacin and ciprofloxacin, resp. For validation, **hair** samples were obtained from six subjects who had been taking one or two of the three fluoroquinolones. Assuming a **hair** growth-rate of 1 cm per mo fluoroquinolones could be detected in the **hair** section(s) that had grown approx. between the dates of drug administration and **hair** sampling.
TI Simultaneous determination of ofloxacin, norfloxacin and ciprofloxacin in human **hair** by high-performance liquid chromatography and fluorescence detection
SO Journal of Chromatography, B: Biomedical Sciences and Applications (**1994**), 653(2), 187-93
CODEN: JCBBEP; ISSN: 1387-2273
AB A high-performance liquid chromatog. method for the simultaneous determination of ofloxacin, norfloxacin and ciprofloxacin in human **hair** is described. A reversed-phase C18 column and a fluorescence detector with switching fluorescence wavelengths were used together with solid-phase extraction of the drugs from **hair** dissolved in 1 M sodium hydroxide. Reproducibility and linearity studies yielded coeffs. of variations of 0.2-2.2, 1.4-3.1 and 1.5-3.4%, and correlation coeffs. of 1.000, 0.999 and 0.999 within the concentration range 0.3-100 ng/mL for ofloxacin, norfloxacin and ciprofloxacin, resp. For validation, **hair** samples were obtained from six subjects who had been taking one or two of the three fluoroquinolones. Assuming a **hair** growth-rate of 1 cm per mo fluoroquinolones could be detected in the **hair** section(s) that had grown approx. between the dates of drug administration and **hair** sampling.
ST **hair** ciprofloxacin norfloxacin ofloxacin HPLC; liq chromatog ciprofloxacin norfloxacin ofloxacin **hair**

IT **Hair**
(ciprofloxacin and norfloxacin and ofloxacin determination in human, by HPLC with fluorescence detection)
IT Chromatography, column and liquid
(high-performance, of ciprofloxacin and norfloxacin and ofloxacin in human **hair**, with fluorescence detection)
IT 13721-01-2D, derivs. 70458-96-7, Norfloxacin **82419-36-1**, Ofloxacin 85721-33-1, Ciprofloxacin
RL: ANT (Analyte); ANST (Analytical study)
(determination of, in human **hair** by HPLC with fluorescence detection)

L123 ANSWER 28 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:633699 HCAPLUS
DOCUMENT NUMBER: 119:233699
TITLE: **Hair** preparations containing levodopa
INVENTOR(S): Rizzo, Antonio
PATENT ASSIGNEE(S): Spain
SOURCE: Eur. Pat. Appl., 6 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 565010	A1	19931013	EP 1993-105555	19930403 <--
R: DE, ES, FR				

PRIORITY APPLN. INFO.: IT 1992-PN30 19920410 <--
AB **Hair** prepns. for stimulation of new **hair** growth, reinvigoration of existing **hair**, and promotion of **hair** repigmentation, comprises levodopa as an active substance and further contains a phosphoric acid salt to strengthen the activation of the local microcirculation, a decarboxylase inhibitor to prevent the composition from spoiling, and a deoxycholic acid to remove the excess of scalp sebum. A **hair** lotion containing levodopa 2.5, creatine phosphate 0.5, ursodeoxycholic acid 0.6, ascorbic acid 0.12g, fragrance q.s., and EtOH/water to 100 mL., is claimed.
TI **Hair** preparations containing levodopa
PI EP 565010 A1 **19931013**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI EP 565010	A1	19931013	EP 1993-105555	19930403 <--
R: DE, ES, FR				

PRAI IT 1992-PN30 19920410 <--
AB **Hair** prepns. for stimulation of new **hair** growth, reinvigoration of existing **hair**, and promotion of **hair** repigmentation, comprises levodopa as an active substance and further contains a phosphoric acid salt to strengthen the activation of the local microcirculation, a decarboxylase inhibitor to prevent the composition from spoiling, and a deoxycholic acid to remove the excess of scalp sebum. A **hair** lotion containing levodopa 2.5, creatine phosphate 0.5, ursodeoxycholic acid 0.6, ascorbic acid 0.12g, fragrance q.s., and EtOH/water to 100 mL., is claimed.
ST **hair** tonic levodopa phosphate deoxycholate ascorbate
IT **Hair preparations**
(lotions, levodopa and creatine phosphate and ascorbate and ursodeoxycholate in)
IT **Hair preparations**
(tonics, levodopa and creatine phosphate and ascorbate and

ursodeoxycholate in)
IT 59-92-7, Levodopa, biological studies
RL: BIOL (Biological study)
(**hair** tonics containing)
IT 50-81-7, L-Ascorbic acid, biological studies 67-07-2, Creatine phosphate 83-44-3D, Deoxycholic acid, derivs. **128-13-2**, Ursodeoxycholic acid 7664-38-2D, Phosphoric acid, salts
RL: BIOL (Biological study)
(**hair** tonics containing levodopa and)
IT 9027-22-9, Decarboxylase
RL: USES (Uses)
(**inhibitors**, **hair** tonics containing levodopa and)

L123 ANSWER 29 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1993:97315 HCAPLUS
DOCUMENT NUMBER: 118:97315
TITLE: Analysis of ofloxacin in **hair** as a measure of **hair** growth and as a time marker for **hair** analysis
AUTHOR(S): Miyazawa, Norio; Uematsu, Toshihiko
CORPORATE SOURCE: Sch. Med., Hamamatsu Univ., Hamamatsu, 431-31, Japan
SOURCE: Therapeutic Drug Monitoring (**1992**), 14(6), 525-8
CODEN: TDMODV; ISSN: 0163-4356
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The distribution of ofloxacin (OFLX) along a single **hair** shaft was analyzed in detail for use as an index of **hair** growth and as a time marker for drug anal. in air. A single **hair** obtained from each of seven subjects, who had taken OFLX for 1-4 days (total of 200-1200 mg) 2.7-5.3 mo before **hair** sampling, was cut into 1-cm-long portions successively from its scalp end. OFLX in each **hair** portion was measured by high-performance liquid chromatog. with a fluorescence detector, and the distance from the scalp end of the **hair** portion containing OFLX was determined Then the other 2-cm long segment of **hair**, which had the above-determined distance at its middle, was cut successively into 2-mm-long pieces and OFLX was determined in each piece. This procedure was repeated in a total of three to four **hair** strands collected from one subject. OFLX was observed to distribute only in one to three consecutive 2-mm-long pieces of **hair**, showing no large diffusion of OFLX along the **hair** shaft with time. Therefore, OFLX distribution may serve as a time marker for analyzing other drugs in **hair**. **Hair** growth rate could be thus estimated and ranged from 0.99 to 1.27 cm/mo (1.12 ± 0.11 cm/mo, mean ± SD) among individuals. The intraindividual variability of **hair** growth rate was 4.8-18.1% (10.3 ± 5.1%) as coefficient of variation.
TI Analysis of ofloxacin in **hair** as a measure of **hair** growth and as a time marker for **hair** analysis
SO Therapeutic Drug Monitoring (**1992**), 14(6), 525-8
CODEN: TDMODV; ISSN: 0163-4356
AB The distribution of ofloxacin (OFLX) along a single **hair** shaft was analyzed in detail for use as an index of **hair** growth and as a time marker for drug anal. in air. A single **hair** obtained from each of seven subjects, who had taken OFLX for 1-4 days (total of 200-1200 mg) 2.7-5.3 mo before **hair** sampling, was cut into 1-cm-long portions successively from its scalp end. OFLX in each **hair** portion was measured by high-performance liquid chromatog. with a fluorescence detector, and the distance from the scalp end of the **hair** portion containing OFLX was determined Then the other 2-cm long

segment of **hair**, which had the above-determined distance at its middle, was cut successively into 2-mm-long pieces and OFLX was determined in each piece. This procedure was repeated in a total of three to four **hair** strands collected from one subject. OFLX was observed to distribute only in one to three consecutive 2-mm-long pieces of **hair**, showing no large diffusion of OFLX along the **hair** shaft with time. Therefore, OFLX distribution may serve as a time marker for analyzing other drugs in **hair**. **Hair** growth rate could be thus estimated and ranged from 0.99 to 1.27 cm/mo (1.12 ± 0.11 cm/mo, mean ± SD) among individuals. The intraindividual variability of **hair** growth rate was 4.8-18.1% (10.3 ± 5.1%) as coefficient of variation.

ST ofloxacin detn chromatog **hair** growth; liq chromatog ofloxacin **hair** growth

IT **Hair**
(of ofloxacin determination in, by HPLC in human, as growth marker)

IT **82419-36-1**, Ofloxacin
RL: ANT (Analyte); ANST (Analytical study)
(determination of, by HPLC human, as **hair** growth rate marker)

L123 ANSWER 30 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1992:120306 HCAPLUS
DOCUMENT NUMBER: 116:120306
TITLE: Possible effect of pigment on the pharmacokinetics of ofloxacin and its excretion in **hair**
AUTHOR(S): Uematsu, Toshihiko; Miyazawa, Norio; Okazaki, Osamu; Nakashima, Mitsuyoshi
CORPORATE SOURCE: Sch. Med., Hamamatsu Univ., Hamamatsu, 431-31, Japan
SOURCE: Journal of Pharmaceutical Sciences (**1992**), 81(1), 45-8
CODEN: JPMSAE; ISSN: 0022-3549
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The mechanism of excretion of the antimicrobial drug ofloxacin in human scalp **hair** was investigated. When black and white **hairs** were taken from a patient with grizzled **hair**, who had been treated with ofloxacin, a much larger quantity of the drug was detected in the black **hair**. To elucidate the cause, the ofloxacin (6, 20, and 60 mg/kg/day) was administered twice a day i.p. for 5 wk to albino and pigmented rats, whose backs had been depilated beforehand. In the last week of administration, the time-plasma concentration profile of ofloxacin was determined One week after the last dosing, the newly grown **hair** on the depilated area was collected, and the drug concentration in the **hair** was measured. The concentration in the **hair** of the pigmented rats correlated with the daily dose, area under the plasma concentration curve (AUC), and maximum plasma concentration (Cmax) at steady state, whereas that in the albino rats correlated with the dose and Cmax only, because AUC did not increase linerly with the dose in the albino rats. The drug concentration in the **hair** of the pigmented rats was always much larger than that in the **hair** of the albino ones, although AUC and Cmax did not differ greatly between both groups. Ofloxacin may be excreted in the **hair** in relation to the dose administered. The mechanism of the excretion may be closely linked with the presence of melanin.

TI Possible effect of pigment on the pharmacokinetics of ofloxacin and its excretion in **hair**

SO Journal of Pharmaceutical Sciences (**1992**), 81(1), 45-8
CODEN: JPMSAE; ISSN: 0022-3549

AB The mechanism of excretion of the antimicrobial drug ofloxacin in human

scalp **hair** was investigated. When black and white **hairs** were taken from a patient with grizzled **hair**, who had been treated with ofloxacin, a much larger quantity of the drug was detected in the black **hair.** To elucidate the cause, the ofloxacin (6, 20, and 60 mg/kg/day) was administered twice a day i.p. for 5 wk to albino and pigmented rats, whose backs had been depilated beforehand. In the last week of administration, the time-plasma concentration profile of ofloxacin was determined One week after the last dosing, the newly grown **hair** on the depilated area was collected, and the drug concentration in the **hair** was measured. The concentration in the **hair** of the pigmented rats correlated with the daily dose, area under the plasma concentration curve (AUC), and maximum plasma concentration (Cmax) at steady state, whereas that in the albino rats correlated with the dose and Cmax only, because AUC did not increase linerly with the dose in the albino rats. The drug concentration in the **hair** of the pigmented rats was always much larger than that in the **hair** of the albino ones, although AUC and Cmax did not differ greatly between both groups. Ofloxacin may be excreted in the **hair** in relation to the dose administered. The mechanism of the excretion may be closely linked with the presence of melanin.

ST **hair** melanin ofloxacin pharmacokinetics

IT Melanins
RL: BIOL (Biological study)
(ofloxacin pharmacokinetics in **hair** in relation to presence of)

IT **Hair**
(ofloxacin pharmacokinetics in, melanin presence in relation to)

IT **82419-36-1**
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(pharmacokinetics of, in **hair**, melanin presence in relation to)

L123 ANSWER 31 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1992:35780 HCAPLUS
DOCUMENT NUMBER: 116:35780
TITLE: Ofloxacin in human **hair** determined by high performance liquid chromatography
AUTHOR(S): Miyazawa, N.; Uematsu, T.; Mizuno, A.; Nagashima, S.; Nakashima, M.
CORPORATE SOURCE: Sch. Med., Hamamatsu, Hamamatsu, 431-31, Japan
SOURCE: Forensic Science International (**1991**), 51(1), 65-77
CODEN: FSINDR; ISSN: 0379-0738
DOCUMENT TYPE: Journal
LANGUAGE: English

AB A procedure is presented for quantitating ofloxacin (OFLX) in human scalp **hair** by high-performance liquid chromatog. with a fluorescence detector. An octadecylsilane column was used, and the mobile phase was a mixture of potassium phosphate buffer (pH 2.6) and acetonitrile. The recovery of OFLX was 90.9-93.8% and within- and between-run precisions were 0.35-1.41% and 1.41-5.49% as the coefficient of variation (CV), resp., when 5-50 ng OFLX was added to 1 mg blank **hair.** The calibration curve was linear in the range 0.5-50 ng/tube (0.5 mL). Interference with other quinolone derivs. could be avoided according to the difference in their retention times or fluorescence spectra. Several pieces of **hair** were obtained from each of twelve healthy male volunteers, who had taken OFLX (100, 300, or 900 mg total dose) over a 1-3-day period 2 wk before the **hair** sampling. In all **hair** samples

except one obtained from a volunteer, who had taken the lowest dose, the 2-cm long segments nearest the scalp contained OFLX (5-45 ng/mg **hair**), while the upper segments did not. A highly significant pos. correlation was observed between the total dose and the concentration of OFLX in the 2-cm long **hair** segments. Such a pos. correlation was also revealed in rat **hair** sampled after repeated i.p. administration of OFLX over a 5-wk period. These results suggest that the measurement of OFLX in **hair** by the present method would be useful for testing patient compliance in clin. pharmacol. as well as for application to forensic science.

TI Ofloxacin in human **hair** determined by high performance liquid chromatography

SO Forensic Science International (**1991**), 51(1), 65-77
CODEN: FSINDR; ISSN: 0379-0738

AB A procedure is presented for quantitating ofloxacin (OFLX) in human scalp **hair** by high-performance liquid chromatog. with a fluorescence detector. An octadecylsilane column was used, and the mobile phase was a mixture of potassium phosphate buffer (pH 2.6) and acetonitrile. The recovery of OFLX was 90.9-93.8% and within- and between-run precisions were 0.35-1.41% and 1.41-5.49% as the coefficient of variation (CV), resp., when 5-50 ng OFLX was added to 1 mg blank **hair**. The calibration curve was linear in the range 0.5-50 ng/tube (0.5 mL). Interference with other quinolone derivs. could be avoided according to the difference in their retention times or fluorescence spectra. Several pieces of **hair** were obtained from each of twelve healthy male volunteers, who had taken OFLX (100, 300, or 900 mg total dose) over a 1-3-day period 2 wk before the **hair** sampling. In all **hair** samples except one obtained from a volunteer, who had taken the lowest dose, the 2-cm long segments nearest the scalp contained OFLX (5-45 ng/mg **hair**), while the upper segments did not. A highly significant pos. correlation was observed between the total dose and the concentration of OFLX in the 2-cm long **hair** segments. Such a pos. correlation was also revealed in rat **hair** sampled after repeated i.p. administration of OFLX over a 5-wk period. These results suggest that the measurement of OFLX in **hair** by the present method would be useful for testing patient compliance in clin. pharmacol. as well as for application to forensic science.

ST ofloxacin detn human **hair** forensic

IT Legal chemistry and medicine
(ofloxacin determination in human **hair** in, by liquid chromatog.)

IT **Hair**
(ofloxacin determination in, of human by liquid chromatog.)

IT **82419-36-1**, Ofloxacin
RL: ANT (Analyte); ANST (Analytical study)
(determination of, in human **hair** by liquid chromatog.)

L123 ANSWER 32 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1981:127147 HCAPLUS
DOCUMENT NUMBER: 94:127147
TITLE: Cosmetic agent for treating the **hair** and scalp
PATENT ASSIGNEE(S): Also Laboratori S.a.S. Dr. P. Sorbini e Co., Italy
SOURCE: Austrian, 5 pp.
CODEN: AUXXAK
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
AT 360160	B	19801229	AT 1978-4522	19780621 <--
AT 7804522	A	19800515		

PRIORITY APPLN. INFO.: AT 1978-4522 19780621 <--

AB A cosmetic for treating the **hair** and scalp to **reduce** scaling and **hair** loss contains 0.6-1% by weight chenodeoxycholic acid [474-25-9] or ursodeoxycholic acid [**128-13-2**], or their salts or derivs. and 0.1-0.25% by weight retinoic acid [302-79-4]. The preparation has a pH of approx. 6, and has a base containing glycerol, propylene glycol, and (or) EtOH, with other optional ingredients.

TI Cosmetic agent for treating the **hair** and scalp

PI AT 360160 **19801229**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI AT 360160	B	19801229	AT 1978-4522	19780621 <--
AT 7804522	A	19800515		

PRAI AT 1978-4522 19780621 <--

AB A cosmetic for treating the **hair** and scalp to **reduce** scaling and **hair** loss contains 0.6-1% by weight chenodeoxycholic acid [474-25-9] or ursodeoxycholic acid [**128-13-2**], or their salts or derivs. and 0.1-0.25% by weight retinoic acid [302-79-4]. The preparation has a pH of approx. 6, and has a base containing glycerol, propylene glycol, and (or) EtOH, with other optional ingredients.

ST bile acid retinoate scalp **hair**; **dandruff** bile acid retinoate; **alopecia** bile acid retinoate

IT **Alopecia**
Dandruff
(bile acids and retinoic acid preparation for control of)

IT 302-79-4
RL: BIOL (Biological study)
(**hair** and scalp preparation containing bile acids and)

IT **128-13-2** 474-25-9
RL: BIOL (Biological study)
(**hair** and scalp preparation containing retinoic acid and)

L123 ANSWER 33 OF 48 HCAPLUS COPYRIGHT 2004 ACS on STN
ACCESSION NUMBER: 1978:494892 HCAPLUS
DOCUMENT NUMBER: 89:94892
TITLE: Chemical composition for treatment of the scalp to **prevent** falling **hair**
INVENTOR(S): Sorbini, Paolo
PATENT ASSIGNEE(S): Also Laboratori S.a.S. Dr. P. Sorbini e Co., Italy
SOURCE: Ger. Offen., 8 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 2758484	A1	19780706	DE 1977-2758484	19771228 <--
DE 2758484	C2	19870129		
FR 2375859	A1	19780728	FR 1978-2	19780102 <--
FR 2375859	B1	19830729		
GB 1560461	A	19800206	GB 1978-63	19780103 <--

US 4185099	A	19800122	US 1978-868563	19780110	<--
CH 636265	A	19830531	CH 1978-6949	19780626	<--
AU 528334	B2	19830428	AU 1978-37488	19780627	<--
AU 7837488	A1	19800103			
CA 1106287	A1	19810804	CA 1978-306632	19780630	<--
JP 63001282	B4	19880112	JP 1978-80693	19780703	<--
JP 55009007	A2	19800122			

PRIORITY APPLN. INFO.: IT 1977-19025 19770104 <--

AB Compns. for treatment of the scalp to **prevent hair** loss contain 0.6-1% of a natural surfactant, such as a bile acid, which acts preferentially on fats and especially on cholesterol, 0.10-0.25% of a cell proliferation regulator such as retinoic acid [302-79-4] or provitamin A, and vehicles or other optional ingredients. For example, a composition contained retinoic acid 0.10, chenodeoxycholic acid [474-25-9] 0.70, nicotinamide 0.20, vitamin H1 0.10, glycerol 30 and propylene glycol 30 g with alc. to give 100 g.

TI Chemical composition for treatment of the scalp to **prevent** falling **hair**

PI DE 2758484 **19780706**

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE	
PI DE 2758484	A1	19780706	DE 1977-2758484	19771228	<--
DE 2758484	C2	19870129			
FR 2375859	A1	19780728	FR 1978-2	19780102	<--
FR 2375859	B1	19830729			
GB 1560461	A	19800206	GB 1978-63	19780103	<--
US 4185099	A	19800122	US 1978-868563	19780110	<--
CH 636265	A	19830531	CH 1978-6949	19780626	<--
AU 528334	B2	19830428	AU 1978-37488	19780627	<--
AU 7837488	A1	19800103			
CA 1106287	A1	19810804	CA 1978-306632	19780630	<--
JP 63001282	B4	19880112	JP 1978-80693	19780703	<--
JP 55009007	A2	19800122			

PRAI IT 1977-19025 19770104 <--

AB Compns. for treatment of the scalp to **prevent hair** loss contain 0.6-1% of a natural surfactant, such as a bile acid, which acts preferentially on fats and especially on cholesterol, 0.10-0.25% of a cell proliferation regulator such as retinoic acid [302-79-4] or provitamin A, and vehicles or other optional ingredients. For example, a composition contained retinoic acid 0.10, chenodeoxycholic acid [474-25-9] 0.70, nicotinamide 0.20, vitamin H1 0.10, glycerol 30 and propylene glycol 30 g with alc. to give 100 g.

ST **hair** loss bile acid compn; scalp **conditioner** bile acid; chenodeoxycholate scalp **conditioner**; retinoate chenodeoxycholate **hair** loss; ursodeoxycholate scalp **hair** loss

IT **Scalp**
(bile acids compns. for treatment of, for **hair** loss **prevention**)

IT **Hair preparations**
(for **hair** loss **prevention**, bile acids in)

IT Bile acids
RL: BIOL (Biological study)
(**hair** loss-**preventing** compns. containing)

IT **128-13-2** 302-79-4 474-25-9
RL: BIOL (Biological study)
(**hair** loss-**preventing** compns. containing)

=> d l123 ibib ab hit 34-45

YOU HAVE REQUESTED DATA FROM FILE 'BIOSIS, HCAPLUS, KOSMET' - CONTINUE? (Y)/N:y

L123 ANSWER 34 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2002:112827 BIOSIS
DOCUMENT NUMBER: PREV200200112827
TITLE: Regulation of human hair follicle cell plasticity and proliferation.
AUTHOR(S): Dana, Richard C. [Reprint author]; Kong, D. [Reprint author]; Yang, J. [Reprint author]; Elliott, M.; Patrick, J. [Reprint author]; Suponeva-Dana, E. [Reprint author]
CORPORATE SOURCE: Committee for World Health, 19571 Pauling, Foothill Ranch, CA, 92610, USA
SOURCE: Molecular Biology of the Cell, (Nov, 2001) Vol. 12, No. Supplement, pp. 29a. print.
Meeting Info.: 41st Annual Meeting of the American Society for Cell Biology. Washington DC, USA. December 08-12, 2001. American Society for Cell Biology.
CODEN: MBCEEV. ISSN: 1059-1524.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 30 Jan 2002
Last Updated on STN: 26 Feb 2002
SO Molecular Biology of the Cell, (Nov, 2001) Vol. 12, No. Supplement, pp. 29a. print.
Meeting Info.: 41st Annual Meeting of the American Society for Cell Biology. Washington DC, USA. December 08-12, 2001. American Society for Cell Biology.
CODEN: MBCEEV. ISSN: 1059-1524.
MY **2001.**
IT Major Concepts
Cell Biology; Integumentary System (Chemical Coordination and Homeostasis)
IT Parts, Structures, & Systems of Organisms
hair follicle cells: integumentary system, cultured, differentiation, plasticity, proliferation; melanocytes: integumentary system
IT Chemicals & Biochemicals
BMP6; S100; WNT; fibroblast growth factor; human telomerase reverse transcriptase; neurotrophin3; sonic hedgehog; telomerase
RN 62031-54-3 (fibroblast growth factor)
130939-66-1 (neurotrophin3)
120178-12-3 (telomerase)

L123 ANSWER 35 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:153611 BIOSIS
DOCUMENT NUMBER: PREV199900153611
TITLE: Activation of telomerase and its association with G1-phase of the cell cycle during UVB-induced skin tumorigenesis in SKH-1 hairless mouse.
AUTHOR(S): Balasubramanian, Sivaprakasam; Kim, Ki-Ho; Ahmad, Nihal; Mukhtar, Hasan [Reprint author]
CORPORATE SOURCE: Dep. Dermatol., Case Western Reserve Univ., 11100 Euclid Ave., Cleveland, OH 44106, USA
SOURCE: Oncogene, (Feb. 11, 1999) Vol. 18, No. 6, pp. 1297-1302. print.
CODEN: ONCNES. ISSN: 0950-9232.
DOCUMENT TYPE: Article

LANGUAGE: English
ENTRY DATE: Entered STN: 16 Apr 1999
Last Updated on STN: 16 Apr 1999

AB **Telomerase** is a ribonucleoprotein enzyme that adds hexanucleotide repeats TTAGGG to the ends of chromosomes. **Telomerase** activation is known to play a crucial role in cell-immortalization and carcinogenesis. **Telomerase** is shown to have a correlation with cell cycle progression, which is controlled by the **regulation** of cyclins, cyclin dependent kinases (cdks) and cyclin dependent kinase **inhibitors** (cdkis). Abnormal expression of these **regulatory** molecules may cause alterations in cell cycle with uncontrolled cell growth, a universal feature of neoplasia. Skin cancer is the most prevalent form of cancer in humans and the solar UV radiation is its major cause. Here, we investigated modulation in **telomerase** activity and protein expression of cell cycle **regulatory** molecules during the development of UVB-induced tumors in SKH-1 **hairless** mice. The mice were exposed to 180 mjoules/cm2 UVB radiation, thrice weekly for 24 weeks. The animals were sacrificed at 4 week intervals and the studies were performed in epidermis. **Telomerase** activity was barely detectable in the epidermis of non-irradiated mouse. UVB exposure resulted in a progressive increase in **telomerase** activity starting from the 4th week of exposure. The increased **telomerase** activity either persisted or further increased with the increased exposure. In papillomas and carcinomas the enzyme activity was comparable and was 45-fold higher than in the epidermis of control mice. Western blot analysis showed an **upregulation** in the protein expression of cyclin DI and cyclin E and their **regulatory** subunits cdk4 and cdk2 during the course of UVB exposure and in papillomas and carcinomas. The protein expression of cdk6 and ckis viz. p16/Ink4A, p21/Waf1 and p27/Kip1 did not show any significant change in UVB exposed skin, but significant **upregulation** was observed both in papillomas and carcinomas. The results suggest that **telomerase** activation may be involved in UVB-induced tumorigenesis in mouse skin and that increased **telomerase** activity may be associated with G1 phase of the cell cycle.

SO Oncogene, (Feb. 11, 1999) Vol. 18, No. 6, pp. 1297-1302. print.
CODEN: ONCNES. ISSN: 0950-9232.

AB **Telomerase** is a ribonucleoprotein enzyme that adds hexanucleotide repeats TTAGGG to the ends of chromosomes. **Telomerase** activation is known to play a crucial role in cell-immortalization and carcinogenesis. **Telomerase** is shown to have a correlation with cell cycle progression, which is controlled by the **regulation** of cyclins, cyclin dependent kinases (cdks) and cyclin dependent kinase **inhibitors** (cdkis). Abnormal expression of these **regulatory** molecules may cause alterations in cell cycle with uncontrolled cell growth, a universal feature of neoplasia. Skin cancer is the most prevalent form of cancer in humans and the solar UV radiation is its major cause. Here, we investigated modulation in **telomerase** activity and protein expression of cell cycle **regulatory** molecules during the development of UVB-induced tumors in SKH-1 **hairless** mice. The mice were exposed to 180 mjoules/cm2 UVB radiation, thrice weekly for 24 weeks. The animals were sacrificed at 4 week intervals and the studies were performed in epidermis. **Telomerase** activity was barely detectable in the epidermis of non-irradiated mouse. UVB exposure resulted in a progressive increase in **telomerase** activity starting from the 4th week of exposure. The increased **telomerase** activity either persisted or further increased with the increased exposure. In papillomas and carcinomas the enzyme activity was comparable and was 45-fold higher than

in the epidermis of control mice. Western blot analysis showed an **upregulation** in the protein expression of cyclin DI and cyclin E and their **regulatory** subunits cdk4 and cdk2 during the course of UVB exposure and in papillomas and carcinomas. The protein expression of cdk6 and ckis viz. p16/Ink4A, p21/Waf1 and p27/Kip1 did not show any significant change in UVB exposed skin, but significant **upregulation** was observed both in papillomas and carcinomas. The results suggest that **telomerase** activation may be involved in UVB-induced tumorigenesis in mouse skin and that increased **telomerase** activity may be associated with G1 phase of the cell cycle.

L123 ANSWER 36 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2000:90376 BIOSIS
DOCUMENT NUMBER: PREV200000090376
TITLE: The use of dihydroxyacetone for photoprotection in variegate porphyria.
AUTHOR(S): Asawanonda, Pravit; Oberlender, Steven; Taylor, Charles [Reprint author]
CORPORATE SOURCE: Department of Dermatology, Massachusetts General Hospital, 55 Fruit Street, Bartlett Hall, Room 410, Boston, MA, 02114, USA
SOURCE: International Journal of Dermatology, (Dec., 1999) Vol. 38, No. 12, pp. 916-918. print.
CODEN: IJDEBB. ISSN: 0011-9059.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Mar 2000
Last Updated on STN: 3 Jan 2002

AB A 33-year-old woman presented with complaints of facial scarring, blisters on the dorsal hands, skin fragility, and increased **hair** growth on the temples. She reported that these "scratch marks" had appeared spontaneously for 3 years. She was otherwise healthy and not on any **medication.** On examination, the patient had several 3-4-mm erythematous papules, some with depressed centers, on the dorsal aspects of the hands (Fig. 1) and the face, but no observable milia. In the perioral region, there were numerous depressed pock-like scars. There was no obvious **hypertrichosis.** A punch biopsy specimen obtained from the left forearm revealed an ulcer with acute and chronic inflammation and periodic acid-Schiff (PAS)-positive material around the dermal blood vessels and adnexae (Fig. 2). Direct immunofluorescence analysis revealed linear immunoglobulin G (IgG) (2+) along the epidermal and adnexal basement membrane zone and around the blood vessels (Fig. 3). C3 (2+) was also present at the epidermal basement membrane zone and around papillary dermal vessels. The patient had a positive hepatitis A antibody, but was negative for hepatitis B and C. Complete blood count and liver function tests were within normal limits. Iron, ferritin, and total iron binding capacity levels were all within normal limits. Antinuclear antibody was positive at 1 : 160 with a speckled pattern, but anti-Ro and anti-La were within normal limits. Total plasma **porphyrins** measured 4 (normal ltoreq 1). A 24-h stool **porphyrin** collection showed diffuse elevations as follows: **coproporphyrin** 307.0 (0-50), uroprophyrin 5.00 (0-4), **protoporphyrin** 515.0 (0-105), total stool **porphyrins** 827.0 (0-159). A 24-h urine **porphyrin** collection also revealed elevations of all the metabolites as follows: porphobilinogen 22.4 (0-2.7), **coproporphyrin** 2211.0 (0-155), uroprophyrin 122.1 (3.3-29.5), **heptacarboxylporphyrin** 35.2 (0-6.8), **hexacarboxylporphyrin** 21.3 (0-0.9), **pentacarboxylporphyrin** 120.8 (0-4.7), total urine **porphyrins** 2510.4 (12-190). The

patient's plasma was diluted with phosphate-buffered saline and scanned between 550 and 650 nm at an excitation wavelength of 405 nm. The emission maximum occurred at 630 nm. In spite of a clinical appearance suggestive of porphyria cutanea tarda (PCT), without any history of acute abdomen or neurologic crises, the clinical diagnosis was clearly variegate porphyria (VP), based on the extensive laboratory abnormalities. At the time of diagnosis, the patient was provided with a list of **medications** which may exacerbate the condition and was instructed to practice vigorous sun protection at all times. Later, she started using a self-tanning lotion. She reported an excellent response to the combination of sunscreen and dihydroxyacetone-containing preparation with far fewer eruptions and a markedly increased tolerance to ambient sun exposure.

SO International Journal of Dermatology, (Dec., 1999) Vol. 38, No. 12, pp. 916-918. print.
CODEN: IJDEBB. ISSN: 0011-9059.

AB A 33-year-old woman presented with complaints of facial scarring, blisters on the dorsal hands, skin fragility, and increased **hair** growth on the temples. She reported that these "scratch marks" had appeared spontaneously for 3 years. She was otherwise healthy and not on any **medication.** On examination, the patient had several 3-4-mm erythematous papules, some with depressed centers, on the dorsal aspects of the hands (Fig. 1) and the face, but no observable milia. In the perioral region, there were numerous depressed pock-like scars. There was no obvious **hypertrichosis.** A punch biopsy specimen obtained from the left forearm revealed an ulcer with acute and chronic inflammation and periodic acid-Schiff (PAS)-positive material around the dermal blood vessels and adnexae (Fig. 2). Direct immunofluorescence analysis revealed linear immunoglobulin G (IgG) (2+) along the epidermal and adnexal basement membrane zone and around the blood vessels (Fig. 3). C3 (2+) was also present at the epidermal basement membrane zone and around papillary dermal vessels. The patient had a positive hepatitis A antibody, but was negative for hepatitis B and C. Complete blood count and liver function tests were within normal limits. Iron, ferritin, and total iron binding capacity levels were all within normal limits. Antinuclear antibody was positive at 1 : 160 with a speckled pattern, but anti-Ro and anti-La were within normal limits. Total plasma **porphyrins** measured 4 (normal ltoreq 1). A 24-h stool **porphyrin** collection showed diffuse elevations as follows: **coproporphyrin** 307.0 (0-50), uroprophyrin 5.00 (0-4), **protoporphyrin** 515.0 (0-105), total stool **porphyrins** 827.0 (0-159). A 24-h urine **porphyrin** collection also revealed elevations of all the metabolites as follows: porphobilinogen 22.4 (0-2.7), **coproporphyrin** 2211.0 (0-155), uroprophyrin 122.1 (3.3-29.5), **heptacarboxylporphyrin** 35.2 (0-6.8), **hexacarboxylporphyrin** 21.3 (0-0.9), **pentacarboxylporphyrin** 120.8 (0-4.7), total urine **porphyrins** 2510.4 (12-190). The patient's plasma was diluted with phosphate-buffered saline and scanned between 550 and 650 nm at an excitation wavelength of 405 nm. The emission maximum occurred at 630 nm. In spite of a clinical appearance suggestive of porphyria cutanea tarda (PCT), without any history of acute abdomen or neurologic crises, the clinical diagnosis was clearly variegate porphyria (VP), based on the extensive laboratory abnormalities. At the time of diagnosis, the patient was provided with a list of **medications** which may exacerbate the condition and was instructed to practice vigorous sun protection at all times. Later, she started using a self-tanning lotion. She reported an excellent response to the combination of sunscreen and dihydroxyacetone-containing preparation with far fewer eruptions and a markedly increased tolerance to ambient sun exposure.

L123 ANSWER 37 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:194573 BIOSIS
DOCUMENT NUMBER: PREV199900194573
TITLE: Determination of endogenous porphyrins and the maximal HpD tumor/normal skin ratio in SKH-1 hairless mice by light induced fluorescence spectroscopy.
AUTHOR(S): Bossu, Edwige [Reprint author]; Padilla, Juan Jose; A' Amar, Ousama; Parache, Robert Michel; Notter, Dominique [Reprint author]; Vigneron, Claude [Reprint author]; Guillemin, Francois
CORPORATE SOURCE: Laboratoire d'Hematologie, Physiologie et Biologie Cellulaire, Faculte des Sciences Pharmaceutiques et Biologiques, Universite Henri Poincare-Nancy I, Nancy, France
SOURCE: Artificial Cells Blood Substitutes and Immobilization Biotechnology, (March, 1999) Vol. 27, No. 2, pp. 109-117. print.
ISSN: 1073-1199.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 25 May 1999
Last Updated on STN: 25 May 1999

AB The treatment of skin tumors is an **application** of photochemotherapy (PCT) which involves an initial administration of a photosensitizer (PS) followed by irradiation with a light beam that causes the PS to produce cytotoxic oxygen species within the tumors. As the PS is also present in normal skin, it is necessary to know how it is distributed between the two tissues. In this study, we have used SKH-1 **hairless** mice bearing papillomas or carcinomas chemically induced. The biodistribution of **hematoporphyrin** derivative (HpD) and the tissue autofluorescence measurements were studied by light induced fluorescence spectroscopy. The tumor and normal autofluorescence spectra measured on control mice with papillomas or carcinomas had a very similar shape. However, the principal endogenous **porphyrin** peak at about 630 nm showed a fluorescence signal amplitude 2 (for papilloma) and 1.5 (for carcinoma) -fold higher than the one found for the normal skin. Moreover, the fluorescence intensity of carcinoma spectrum is 1.4-fold lower than the one of papilloma spectrum at 630 nm. The tissue autofluorescence can be used to distinguish tumor from normal skin and benign from malignant tumor. This difference in fluorescence intensity at 630 nm was directly related to the concentration of endogenous **porphyrins** in the tumor. Fluorescence intensity ratios between tumor and normal skin were measured 4, 8, 24, 48, 72 and 96 hours after intraperitoneal injection of HpD (5 mg/kg body weight). The best tumor/normal skin ratio was 6.2 for HpD and the time required to reach this ratio was 48 h. HpD showed a moderate selectivity since the ratio was higher than 1 during the four first days. Photodynamic therapy with the same dose of HpD used in this biodistribution study must also be carried out to verify that the maximal tumor/skin ratio corresponds to the maximal efficiency of HpD.

SO Artificial Cells Blood Substitutes and Immobilization Biotechnology, (March, 1999) Vol. 27, No. 2, pp. 109-117. print.
ISSN: 1073-1199.

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hairless mice bearing papillomas or carcinomas chemically induced. The biodistribution of **hematoporphyrin** derivative (HpD) and the tissue autofluorescence measurements were studied by light induced fluorescence spectroscopy. The tumor and normal autofluorescence spectra measured on control mice with papillomas or carcinomas had a very similar shape. However, the principal endogenous **porphyrin** peak at about 630 nm showed a fluorescence signal amplitude 2 (for papilloma) and 1.5 (for carcinoma) -fold higher than the one found for the normal skin. Moreover, the fluorescence intensity of carcinoma spectrum is 1.4-fold lower than the one of papilloma spectrum at 630 nm. The tissue autofluorescence can be used to distinguish tumor from normal skin and benign from malignant tumor. This difference in fluorescence intensity at 630 nm was directly related to the concentration of endogenous **porphyrins** in the tumor. Fluorescence intensity ratios between tumor and normal skin were measured 4, 8, 24, 48, 72 and 96 hours after intraperitoneal injection of HpD (5 mg/kg body weight). The best tumor/normal skin ratio was 6.2 for HpD and the time required to reach this ratio was 48 h. HpD showed a moderate selectivity since the ratio was higher than 1 during the four first days. Photodynamic therapy with the same dose of HpD used in this biodistribution study must also be carried out to verify that the maximal tumor/skin ratio corresponds to the maximal efficiency of HpD.

L123 ANSWER 38 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:413543 BIOSIS
DOCUMENT NUMBER: PREV199900413543
TITLE: Bladerunners and telomerases.
AUTHOR(S): Hussain, Mehboob A. [Reprint author]
CORPORATE SOURCE: Laboratory of Molecular Endocrinology, Howard Hughes Medical Institute, Massachusetts General Hospital, 50 Blossom Street Wellman 320, Boston, MA, 02114, USA
SOURCE: European Journal of Endocrinology, (Aug., 1999) Vol. 141, No. 2, pp. 98-100. print.
ISSN: 0804-4643.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 18 Oct 1999
Last Updated on STN: 18 Oct 1999
SO European Journal of Endocrinology, (Aug., 1999) Vol. 141, No. 2, pp. 98-100. print.
ISSN: 0804-4643.
IT Major Concepts
Aging; Enzymology (Biochemistry and Molecular Biophysics)
IT Parts, Structures, & Systems of Organisms
bone marrow: blood and lymphatics, immune system; gastrointestinal crypt cells: digestive system; germ cell: reproductive system; **hair follicles: integumentary system**; liver: digestive system; neural tube: nervous system; skin fibroblast cells: integumentary system; somatic cells; splenocytes: blood and lymphatics; stem cell; telomere
IT Diseases
alopecia: integumentary system disease
Alopecia (MeSH)
IT Diseases
dermal fibrosis: integumentary system disease
IT Diseases
epidermal hyperplasia: integumentary system disease
IT Diseases
hyperkeratosis: integumentary system disease
Keratosis (MeSH)

IT Chemicals & Biochemicals
telomerase: expression; DNA polymerase
RN **120178-12-3** (telomerase)
9012-90-2 (DNA polymerase)

L123 ANSWER 39 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1998:257810 BIOSIS
DOCUMENT NUMBER: PREV199800257810
TITLE: In situ hybridization analysis of the expression of human telomerase RNA in normal and pathologic conditions of the skin.
AUTHOR(S): Ogoshi, Machiko; Le, Thuy; Shay, Jerry W.; Taylor, R. Stan [Reprint author]
CORPORATE SOURCE: Dep. Dermatol., Univ. Texas Southwestern Med. Cent., 5323 Harry Hines Blvd., Dallas, TX 75235, USA
SOURCE: Journal of Investigative Dermatology, (May, 1998) Vol. 110, No. 5, pp. 818-823. print.
CODEN: JIDEAE. ISSN: 0022-202X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 9 Jun 1998
Last Updated on STN: 12 Aug 1998

AB Human telomerase RNA (hTER) expression in skin was examined by in situ hybridization analysis. AR newborn foreskins examined (n=5) expressed hTER in epidermal basal cells at moderate levels. Telomerase RNA was not detectable in most adult specimens from sun protected areas (six of seven), whereas all samples obtained from sun exposed areas (n=8) showed moderate hTER signals in epidermal basal cells. Telomerase RNA was also detected at moderate to strong levels in basal cells of psoriasis, contact dermatitis, and the proliferative cells of the anagen hair bulb. Basal cell carcinoma samples (14 of 15) had moderate to high hTER expression throughout the entire tumor, whereas squamous cell carcinomas (seven of eight) showed variable intensities of hTER expression but only in the cells at the periphery of tumor nests. All 'melanomas examined (n=5) had moderate hTER expression in all tumor cells. The hTER signal intensities in skin tumors did not correlate with the age or sex of the donors, the clinical history of the lesions, or the histologic subtypes. To address whether hTER expression correlated with the proliferative state, sequential sections were stained with anti-Ki-67 antibody, a proliferation marker. In newborn foreskins, squamous cell carcinomas, and basal cell carcinomas, the distributions of hTER and Ki-67 were similar but not always identical. Telomerase RNA was more abundant than Ki67 in the basal and suprabasal layer of newborn foreskins, suggesting that hTER expression is present both in actively cycling and in resting cells.

SO Journal of Investigative Dermatology, (May, 1998) Vol. 110, No. 5, pp. 818-823. print.
CODEN: JIDEAE. ISSN: 0022-202X.

IT Major Concepts
Integumentary System (Chemical Coordination and Homeostasis); Molecular Genetics (Biochemistry and Molecular Biophysics)
IT Parts, Structures, & Systems of Organisms
anagen **hair** bulb: integumentary system; epidermal basal cells: integumentary system; foreskin: integumentary system; skin: integumentary system
IT Diseases
basal cell carcinoma: integumentary system disease, neoplastic disease
Carcinoma, Basal Cell (MeSH)
IT Diseases
contact dermatitis: integumentary system disease
Dermatitis, Contact (MeSH)

IT Diseases
melanoma: neoplastic disease
Melanoma (MeSH)
IT Diseases
psoriasis: integumentary system disease
Psoriasis (MeSH)
IT Diseases
squamous cell carcinoma: neoplastic disease
Carcinoma, Squamous Cell (MeSH)
IT Chemicals & Biochemicals
telomerase RNA: expression; Ki-67
RN **120178-12-3** (TELOMERASE)

L123 ANSWER 40 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1998:478107 BIOSIS
DOCUMENT NUMBER: PREV199800478107
TITLE: The evolution of aging: A new approach to an old problem of biology.
AUTHOR(S): Bowles, J. T. [Reprint author]
CORPORATE SOURCE: 925 West Huron No. 407, Chicago, IL 60622, USA
SOURCE: Medical Hypotheses, (Sept., 1998) Vol. 51, No. 3, pp. 179-221. print.
CODEN: MEHYDY. ISSN: 0306-9877.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 5 Nov 1998
Last Updated on STN: 5 Nov 1998

AB Most gerontologists believe aging did not evolve, is accidental, and is unrelated to development. The opposite viewpoint is most likely correct. Genetic drift occurs in finite populations and leads to homozygosity in multiple-alleled traits. Episodic selection **events** will alter random drift towards homozygosity in alleles that increase fitness with respect to the selection **event.** Aging increases population turnover, which accelerates the benefit of genetic drift. This advantage of aging led to the evolution of aging systems (ASs). Periodic predation was the most prevalent episodic selection pressure in evolution. Effective defenses to predation that allow exceptionally long lifespans to evolve are shells, extreme intelligence, isolation, and flight. Without episodic predation, aging provides no advantage and aging systems will be deactivated to increase reproductive potential in unrestricted environments. The periodic advantage of aging led to the periodic evolution of aging systems. Newer aging systems co-opted and added to prior aging systems. Aging organisms should have one dominant, aging system that co-opts vestiges of earlier-evolved systems as well as vestiges of prior systems. In human evolution, aging systems chronologically emerged as follows: telomere shortening, mitochondrial aging, mutation accumulation, senescent gene expression (AS4), targeted somatic tissue apoptotic-atrophy (AS5), and female reproductive tissue apoptotic-atrophy (AS6). During famine or drought, to avoid extinction, reproduction is curtailed and aging is **slowed** or somewhat reversed to postpone or reverse reproductive senescence. AS4-AS6 are gradual and reversible aging systems. The life-extending/rejuvenating effects of caloric restriction support the idea of aging reversibility. Development and aging are timed by the gradual loss of cytosine methylation in the genome. Methylated cytosines (5mC) **inhibit** gene transcription, and deoxyribonucleic acid (DNA) cleavage by restriction enzymes. Cleavage **inhibition prevents** apoptosis, which requires DNA fragmentation. Free radicals catalyze the demethylation of 5mC while antioxidants catalyze the remethylation of cytosine by altering the activity of DNA methyltransferases. Hormones act

as either surrogate free radicals by stimulating the cyclic adenosine monophosphate (cAMP) pathway or as surrogate antioxidants through cyclic guanosine monophosphate (cGMP) pathway stimulation. Access to DNA containing 5mC **inhibited** developmental and aging genes and restriction sites is allowed by DNA helicase strand separation. Tightly wound DNA does not allow this access. The DNA helicase generates free radicals during strand separation; hormones either amplify or counteract this effect. Caloric restriction **slows** or reverses the aging process by increasing melatonin levels, which suppresses reproductive and free radical hormones, while increasing antioxidant hormone levels. Cell apoptosis during CR leads to somatic wasting and a release of DNA, which increases bioavailable cGMP. The rapid aging diseases of progeria, the three diseases: (xeroderma pigmentosum (XP), Cockayne syndrome(CS), and ataxia telangiectasia (AT)), and Werner's syndrome are related to or caused by defects in three separate DNA helicases. The rapid aging diseases caused by mitochondrial malfunctions mirror those seen in XP, CS, and AT. Comparing these diseases allows for assignment of the different symptoms of aging to their respective aging systems. **Follicle** -stimulating hormone (FSH) demethylates the genes of AS4, luteinizing hormone (LH) of AS5, and estrogen of AS6 while cortisol may act cooperatively with FSH and LH, and 5-alpha dihydrotestosterone (DHT) with FSH in these role. The Werner's DNA helicase links timing of the age of puberty, menopause, and maximum lifespan in one mechanism. **Telomerase** is under hormonal control. Most cancers likely result from malfunctions in the programmed apoptosis of AS5 and AS6. The Hayflick limit is reached primarily through loss of cytosine methylation of genes that **inhibit** replication. Men suffer the diseases of AS4 at a higher rate than women who suffer from AS5 more often. Adult mammal cloning suggests aging-related cellular demethylation, and thus aging, is reversible. This theory suggests that the protective effect of smoking and ibuprofen for Alzheimer's disease is caused through LH suppression.

SO Medical Hypotheses, (Sept., 1998) Vol. 51, No. 3, pp. 179-221. print. CODEN: MEHYDY. ISSN: 0306-9877.

AB Most gerontologists believe aging did not evolve, is accidental, and is unrelated to development. The opposite viewpoint is most likely correct. Genetic drift occurs in finite populations and leads to homozygosity in multiple-alleled traits. Episodic selection **events** will alter random drift towards homozygosity in alleles that increase fitness with respect to the selection **event.** Aging increases population turnover, which accelerates the benefit of genetic drift. This advantage of aging led to the evolution of aging systems (ASs). Periodic predation was the most prevalent episodic selection pressure in evolution. Effective defenses to predation that allow exceptionally long lifespans to evolve are shells, extreme intelligence, isolation, and flight. Without episodic predation, aging provides no advantage and aging systems will be deactivated to increase reproductive potential in unrestricted environments. The periodic advantage of aging led to the periodic evolution of aging systems. Newer aging systems co-opted and added to prior aging systems. Aging organisms should have one dominant, aging system that co-opts vestiges of earlier-evolved systems as well as vestiges of prior systems. In human evolution, aging systems chronologically emerged as follows: telomere shortening, mitochondrial aging, mutation accumulation, senescent gene expression (AS4), targeted somatic tissue apoptotic-atrophy (AS5), and female reproductive tissue apoptotic-atrophy (AS6). During famine or drought, to avoid extinction, reproduction is curtailed and aging is **slowed** or somewhat reversed to postpone or reverse reproductive senescence. AS4-AS6 are gradual and reversible aging systems. The life-extending/rejuvenating effects of caloric restriction support the idea of aging reversibility.

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L123 ANSWER 41 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1999:40323 BIOSIS
DOCUMENT NUMBER: PREV199900040323
TITLE: The effect of vitamin E acetate on ultraviolet-induced mouse skin carcinogenesis.
Berton, Thomas R.; Conti, Claudio J.; Mitchell, David L.; Aldaz, C. Marcelo; Lubet, Ronald A.; Fischer, Susan M. [Reprint author]
Univ. Texas M.D. Anderson Cancer Center, Sci. Park-Res. Div., P.O. Box 389, Smithville, TX 78957, USA
Molecular Carcinogenesis, (Nov., 1998) Vol. 23, No. 3, pp. 175-184. print.
CODEN: MOCAE8. ISSN: 0899-1987.
Article
English
ENTRY DATE: Entered STN: 3 Feb 1999
Last Updated on STN: 3 Feb 1999

AB Despite the benefits of sunscreens, ultraviolet (UV) exposure can still lead to skin cancer. in this study we investigated the effect of topical application of the antioxidant vitamin E acetate (VEA) on the

inhibition of UV-induced carcinogenesis. **Hairless** SKH-1 mice received 5.2 mg of VEA 30 min before (VEA/UV) or after (UV/ VEA) a single minimal erythemic dose of UV light. Vehicle-control animals received acetone 30 min before UV exposure (Ace/UV). After 24 h, cyclobutane dimer repair was twofold and 1.5-fold greater in the UV/VEA and VEA/UV groups, respectively. Expression of p53 protein in the UV/V/VEA group was maximum at 12 h after UV exposure, whereas in the Ace/UV- and VEA/UV-treated mice, maximum p53 immunostaining was statistically higher at 15 h (P = 0.03). DNA synthesis as determined by 5-bromo-2'-deoxyuridine incorporation was twofold higher after 15 h in all groups but was not statistically different among treatment groups. Protein levels of cyclin D1 and p21 were increased in both VEA groups by 6 h. In addition, VEA treatments delayed tumor formation and yield for the first 20 wk, although this difference was lost by 30 wk. The **telomerase** activity of carcinomas from the UV/EA-treated mice was statistically lower than that of the Ace/UV-treated mice (P = 0.05). This study showed that although VEA may mitigate some of the initial **events** associated with UV irradiation such as DNA damage and p53 expression, it has limited potential in **preventing** UV-induced proliferation and tumor formation.

SO Molecular Carcinogenesis, (Nov., 1998) Vol. 23, No. 3, pp. 175-184. print. CODEN: MOCAE8. ISSN: 0899-1987.

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L123 ANSWER 42 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 1997:234470 BIOSIS

DOCUMENT NUMBER: PREV199799533673

TITLE: Molecular cloning of functional human telomerase RNA component promoter: Regulation of telomerase activity in human keratinocyte.

AUTHOR(S): Kallassy, M.; Souabni, A.; Martel, N.; Nakazawa, H.

CORPORATE SOURCE: Int. Agency Res. Cancer/World Health Organization, Lyon F 69372, France

SOURCE: Proceedings of the American Association for Cancer Research Annual Meeting, (1997) Vol. 38, No. 0, pp. 637. Meeting Info.: Eighty-eighth Annual Meeting of the American Association for Cancer Research. San Diego, California, USA. April 12-16, 1997.

ISSN: 0197-016X.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 2 Jun 1997
Last Updated on STN: 2 Jun 1997
SO Proceedings of the American Association for Cancer Research Annual Meeting, (1997) Vol. 38, No. 0, pp. 637.
Meeting Info.: Eighty-eighth Annual Meeting of the American Association for Cancer Research. San Diego, California, USA. April 12-16, 1997.
ISSN: 0197-016X.
MY **1997.**
IT Miscellaneous Descriptors
ACTIVITY; ENZYMOLOGY; **HAIR FOLLICLE**; HUMAN **TELOMERASE** RNA COMPONENT; INTEGUMENTARY SYSTEM; KERATINOCYTE; **TELOMERASE**; **TELOMERASE REGULATOR**

L123 ANSWER 43 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1998:220527 BIOSIS
DOCUMENT NUMBER: PREV199800220527
TITLE: Experimental model for porphyria cutanea tarda induced by hexachlorobenzene in hairless mice.
AUTHOR(S): Federico, M. L.; Schaller, M. V.; Fukuda, H.; Stella, A. M.; Batlle, A. M. Del C.
CORPORATE SOURCE: Dep. Quim. Biol., Fac. Ciencias Exactas Nat., Univ. Buenos Aires, Buenos Aires, Argentina
SOURCE: Revista Argentina de Dermatologia, (July-Sept., 1997) Vol. 78, No. 3, pp. 137-148. print.
CODEN: RADEBD. ISSN: 0325-2787.
DOCUMENT TYPE: Article
LANGUAGE: Spanish
ENTRY DATE: Entered STN: 11 May 1998
Last Updated on STN: 11 May 1998
AB In this paper we describe the effect of HCB in **hairless** mice in order to obtain an experimental model of PCT. This model would be useful to study the cutaneous photosensitivity of the disease and to assay dermatologic creams. HCB is a polyhalogenated hydrocarbon which has been successfully used in Balb/C and C57 BL strain mice. Animals received one single dose of 200 mg HCB/kg (i.p.) three days after one injection of 12.5 mg Fe (i.p.). Two groups of controls were used: one of them without any treatment and the other with iron pretreatment only. At different times the animals were sacrificed and **porphyrin** content in skin, liver, urine and feces were determined. In addition the hepatic Uro-D activity, Cyt-P450 and lipid peroxidation were measured, together with liver and skin glutathione concentration. In both sexes, we observed a 40% increase in the liver/body weight ratio in the intoxicated groups as well as in iron controls. **Porphyrin** levels increased in liver and skin; such increase occurred after a reduction in glutathione levels. On the contrary, in males the **porphyrin** levels raised only in skin, while glutathione levels increased in both tissues. The activity of hepatic Uro-D decreased 70% in females without recovering to normal value even at day 31 (Control S.A. = 0.32 U/mg). In the intoxicated animals, both females and males, an increase in the levels of hepatic Cyt-P450 and lipid peroxidation was observed. These results showed that although we could not obtain a clear **porphyrinogenic** response to HCB, we reproduce the first steps of the PCT, being necessary to adjust the **intoxication** protocol. However, we cannot discard the possibility that the **hairless** strain is not susceptible to the prophyrinogenic affect of HCB. In this case we could only reproduce the cutaneous symptomatology through other methods such as **porphyrin**

or delta aminolevulic acid topically applied to skin.

SO Revista Argentina de Dermatologia, (July-Sept., 1997) Vol. 78, No. 3, pp. 137-148. print.
CODEN: RADEBD. ISSN: 0325-2787.

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L123 ANSWER 44 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1997:66449 BIOSIS
DOCUMENT NUMBER: PREV199799365652
TITLE: Telomerase activity concentrates in the mitotically active segments of human hair follicles.
AUTHOR(S): Ramirez, Ruben D.; Wright, Woodring E.; Shay, Jerry W.; Taylor, R. Stan [Reprint author]
CORPORATE SOURCE: Dep. Dermatology, Univ. Texas Southwestern Med. Cent., 5323 Harry Hines Boulevard, Dallas, TX 75235-9069, USA
SOURCE: Journal of Investigative Dermatology, (1997) Vol. 108, No. 1, pp. 113-117.
CODEN: JIDEAE. ISSN: 0022-202X.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 11 Feb 1997
Last Updated on STN: 11 Feb 1997

AB **Telomerase** is a ribonucleoprotein enzyme capable of adding hexanucleotide repeats onto the ends of linear chromosomal DNA. Whereas normal somatic cells with a limited replicative capacity fail to express **telomerase** activity, most immortal eukaryotic cells do. Cells of renewal tissues (e.g., skin, intestine, blood) require an extensive proliferative capacity. Some cells in such renewal tissues also express **telomerase** activity, most likely to **prevent** rapid erosion of their telomeres during cell proliferation. In this study. we measured the levels of **telomerase** activity in dissected compartments of the human **hair follicle: hair** shaft, gland-containing fragment, upper intermediate fragment (where it is

thought undifferentiated stem cells reside), lower intermediate fragment, and in the bulb-containing fragment (an area with high mitotic activity containing a more differentiated pool of keratinocytes). In anagen **follicles**, high levels of **telomerase** activity were found almost exclusively in the bulb-containing fragment of the **follicles**, with low levels of **telomerase** in the bulge area (intermediate fragments) and gland-containing fragment. In comparison, catagen **follicles** had low levels of **telomerase** activity in the bulb-containing fragments as well as in other compartments. Such observations indicate that, in anagen **hair follicles**, the fragments containing cells actively dividing (e.g., transient amplifying cells) express **telomerase** activity, whereas fragments containing cells with low mitotic activity, for example, quiescent stem cells, express low levels of **telomerase** activity.

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L123 ANSWER 45 OF 48 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1984:184117 BIOSIS
DOCUMENT NUMBER: PREV198477017101; BA77:17101
TITLE: COMPARATIVE ACTIVITY OF BENZOYL PER OXIDE AND HEXACHLOROPHENE IN-VIVO STUDIES AGAINST PROPIONIBACTERIUM-ACNES IN HUMANS.
AUTHOR(S): NACHT S [Reprint author]; GANS E H; MCGINLEY K J; KLIGMAN A M
CORPORATE SOURCE: VICKS RES CENT, 1 FAR MILL CROSSING, SHELTON, CT 06484, USA
SOURCE: Archives of Dermatology, (1983) Vol. 119, No. 7, pp. 577-579.
CODEN: ARDEAC. ISSN: 0003-987X.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH

AB The bactericidal effects of benzoyl peroxide (5% lotion) and

hexachlorophene (3% colloidal suspension) against P. acnes were compared in 9 healthy college students who had the microbiological and skin lipid characteristics typical of acne vulgaris, but no active lesions. Each of the 2 **medications** was applied twice daily to opposite sides of the face for 4 consecutive weeks. Hexachlorophene was effective against surface aerobes, but only slightly active against P. acnes. It marginally reduced free fatty acid concentrations in surface lipids and in follicular **porphyrin** fluorescence. Benzoyl peroxide virtually eliminated P. acnes and aerobes and induced substantially decreased free fatty acid concentrations and follicular fluorescence. Thus, benzoyl peroxide exerts its antimicrobial action in the **follicles** and inhibits P. acnes; the antimicrobial effectiveness of hexachlorophene is limited to the skin surface.

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YOU HAVE REQUESTED DATA FROM FILE 'BIOSIS, HCAPLUS, KOSMET' - CONTINUE? (Y)/N:y

YOU HAVE REQUESTED DATA FROM 3 ANSWERS - CONTINUE? Y/(N):y

L123 ANSWER 46 OF 48 KOSMET COPYRIGHT 2004 IFSCC on STN
ACCESSION NUMBER: 28649 KOSMET
FILE SEGMENT: scientific, technical
TITLE: RHAMNOSE-RICH AND FUCOSE-RICH OLIGO- AND POLYSACCHARIDES (RROP-S AND FROPS), AGONISTS AND ANTAGONISTS OF CELL-MEMBRANE RECEPTORS AS NEW ACTIVE PRINCIPLES AGAINST SKIN AGING
AUTHOR: ROBERT L (ROBERT L (1), ROBERT AM (1), GESZTESI JL (2), LUPPI E (2)=UNIVERSITY PARIS 6 AND HOTEL DIEU PARIS AND [illegible]TITUT DERM, PARIS, FRANCE (1), NATURA [illegible]O, BRAZIL (2)); ROBERT AM; GESZTESI JL;
SOURCE: [illegible]CE 2003, SEOUL, KOREA, SEPTEMBER 22-24, [illegible]VENTION CENTRE, SEOUL, CONFERENCE THEME: [illegible]RE SCIENCE MEETS DREAM, PROCEEDINGS [illegible]PER 26, 352-373, 40 REFS [illegible]er: SOCIETY OF COSMETIC SCIENTISTS OF [illegible]14-1, BORA-RI, KIHEUNG-EUP, YONGIN-SI [illegible]729, KOREA, TEL: +82-31-280 57 01, FAX: [illegible]24, EMAIL: Changkim@pacific.co.kr , INTERNET: www.scsk.or.kr ; IFSCC / SOCIETY OF COSMETIC SCIENTISTS, GT HOUSE, 24-26 ROTHESAY ROAD, LUTON, BEDS LU1 1QX, UNITED KINGDOM, TEL: +44-1582-726661, FAX:

+44-1582-405217, EMAIL: ifscc.scs@btinternet.com
Availability: SOCIETY OF COSMETIC SCIENTISTS OF KOREA (SCSK), 314-1, BORA-RI, KIHEUNG-EUP, YONGIN-SI KYUNGGI-DO 449-729, KOREA, TEL: +82-31-280 57 01, FAX: +82-31-285 03 24, EMAIL: Changkim@pacific.co.kr , INTERNET: www.scsk.or.kr

DOCUMENT TYPE: Conference
LANGUAGE: English

AB Rhamnose-rich (RROP-s) and fucose-rich (FROP-s) oligo- and polysaccharides were prepared and extensively characterised by physical and chemical procedures and compared to L-fucose. Their biological properties were then studied on human skin fibroblast cell cultures, human skin explant cultures and on hairless rat skin, using a variety of cell-biological, biochemical and computerised morphometrical procedures. Among the most important properties we could establish, the following are of particular interest for the tretment and **prevention** of age-dependent modifications of human skin (loss of skin-tissue, cells and matrix, wrinkle formation and others) : stimulation of cell proliferation (by 3[H]-thymidine incorporation and the MTT test), scavenging of reactive oxygen species (ROS) using several different procedures, and protease (MMP-2 and MMP-9) down-**regulation.** A topical preparation, using RROP-s and FROP-s, and/or L-fucose, was shown to increase cell proliferation, dermal matrix synthesis, efficient scavenging of ROS-s and to increase also the thickness of dermal tissue when applied for 4 weeks on hairless rat skin, accompanied by the densification of collagen bundles as well as by an increase of elastin synthesis. Using fluorescent labeled FROPs, it could be shown that these oligosaccharides react with cell-membrane receptors and especially with the elastin-laminin-receptor and the fucose-mannose receptor, but they penetrate also in the cell nucleus, suggesting the possibility of a direct action on the **regulation** of gene expression. When applied to the human skin of a team of voluntary women encompassing all age-groups, the efficiency of FROP-containing preparation could be confirmed using indentometry and computerised evaluation of skin micro-relief, as well as evaluation of periorbital wrinkles. It appears therefore that these preparations correspond to all the requirements of active anti-aging principles. Skin aging became an important issue of our rapidly aging society. 20 to 35% of the general population in all advanced and in most advancing countries is above 65 years. The most rapidly increasing fraction of this population are the very old (centenarians). This fact creates an important market for anti-aging skin products addressing an increasingly exigent population. Modern research carried out in some laboratories is taking up this challenge with increasing efficiency. Before describing the results obtained in our laboratory along these lines of investigation, we have to discuss in some detail the basic cellular and molecular mechanisms involved in skin aging. In order to define the most significant tests for screening studies, the first important issue is to define the mechanisms which underly skin aging. Aging of tissues is a complex process which involves the aging of cells, of extracellular matrix (ECM) and of cell-matrix interactions. These interactions are mediated by receptors as the integrins or the elastin-laminine-receptor (ELR). Both types of receptors (as well as some others) are involved in the aging process. Cell-aging, as studied in cell cultures, follows the principles elaborated by L. Hayflick and is explained essentially by the telomer-**telomerase**-centered mechanisms. Cell aging affects cell divisions both for keratinocytes and fibroblasts which is progressively **slowing** down with age. As shown however by the **slow** but efficient wound healing of elderly subjects, it remains sufficient to close surgical or accidental wounds. An other important aspect of cell

aging is the progressive modification of the 'program' of the biosynthesis of ECM components. This is an important aspect of skin aging because of the rich ECM of dermis. Loss of dermal tissue measured as the skin thickness of biopsy specimens from sun-protected sites gave an average estimate of 7% of the original skin thickness (extrapolated to birth) lost every 10 years. This leaves less than the one third of the original skin thickness at about 90 - 100 years. This loss of skin tissue is the combined result of cell loss and mainly of **reduced** cell-biosynthetic activity, as well as of increased proteolytic activity. Age-dependent loss of collagen is the result of the combined effect of decreased biosynthesis and increased degradation, as the result of the age-dependent increase of the local production of matrix degrading enzymes. As shown on Fig.2., elastase-type endopeptidase activity is steadily increasing with chronological age and also during in vitro aging, with increasing cell passages as shown with human skin fibroblasts. Decreasing matrix biosynthetic activity combined with increasing matrix degradation are the two essential ingredients of skin aging. Besides proteolytic enzymes, reactive oxygen species comprising free radicals as hydroxyl radical, superoxide and hydrogen peroxide represent another important source of skin degrading agents. ROS-production is both an intrinsic, cell-dependent process and also a photochemically, UV-induced mechanism. It was shown however, that UVA-induced free radical production was much more important than UVB-induced production, is maximal at the skin surface and decreases rapidly towards the dermis. The metabolic generation of ROS is however cell-dependent, essentially of mitochondrial origin, and was shown to increase with age, together with a decrease of the cellular scavenging activity. We could show that hyaluronan, one of the most important glycosaminoglycan components of skin, is highly sensitive to free radical degradation. This reaction could be used for the quantitative determination of free radical generation. Hyaluronan is produced both in the dermis and epidermis and is involved in a number of important biological properties of skin tissue, such as hydration, control of molecular traffic, activation of MMP-2 and MMP-9 and others. Bedsides these mechanisms concerning matrix production and matrix degradation, there is another important aspect of skin homeostasis, the fine adjustment of the relative rates of the expression of genes coding for the ECM-components, collagens, elastin and others. In this respect receptor-mediated cell-matrix interactions play a crucial role. This receptor-mediated information exchange between the cells and the surrounding ECM-components is progressively deteriorating with age, as we could demonstrate in our experiments with the elastin-laminin-receptor. In cells from old individuals (>65 years) this receptor appeared to be uncoupled from its normal transmission pathway as established on circulating white blood cells and endothelial cells or fibroblasts. One of the most conspicuous results of this uncoupling of ELR is the loss of its coupling to the Gi -component of its transmission pathway, accompanied however by an increased free radical production. This can easily damage the cell-membrane and account for the loss of the calcium homeostatic **regulations** of the cells, demonstrated experimentally on PMN-leucocytes obtained from aged-pathological donors. The above summarised mechanisms, cell proliferation, matrix production and degradation, ROS-scavenging are the reactions we explored systematically for the characterisation of new active principles. This is a somewhat abusive (but largely used) term. It would be more appropriate to speak about the **slowing** down of aging processes. The demonstration of "peace-meal" aging of tissue functions ("vieillissement en pieces detaches" in French) shows clearly that different tissues and functions age at different rates. Some functions decrease rapidly with age, others much more **slowly.** Articular cartilage looses

rapidly its biomechanical characteristics, most elastic functions as accomodation of the eye lens, elasticity of blood vessels , of the lung or of the skin decline also relatively rapidly. Other functions, related essentially to the central nervous system, decline more **slowly.** This is true also for the skin, some of its components decrease rapidly (skin collagen and glycosaminoglycans), others may even increase with age, as fibronectine. The result is a progressively changing macromolecular composition of skin matrix as demonstrated also by the age-dependent modifications of its rheological properties. All the above described factors were taken in consideration for the elaboration of some new active principles designed to counteract the above described mechanisms underlying skin aging. In the present experiments we tested rhamnose- and fucose-rich oligo- and polysaccharides (RROPs and FROPs). The biological origin and chemical preparation and characterisation of these substances was recently described. Here we shall concentrate on the biological-biochemical characteristics of these substances in relation to the above described aging mechanisms. The above described and succently summarised favourable results obtained with the oligo- polysaccharide preparations, designated RROP-s and FROP-s, need to be explained in terms of mechanisms of action at the level of the cellular and molecular components of the skin. We could show with fluorescent-labelled FROP-preparations, that they have two major sites of interaction with human skin fibroblasts: the cell membrane and the nucleus. Interaction with the cell membrane is maintained even for formol-fixed cells, but nuclear penetration is suppressed. Interaction of FROP-s with cell membrane components appears to concern two types of receptors: the elastin-laminin receptor and the fucose-mannose receptor. The presence of a specific alpha-L-rhamnose recognising receptor was demonstrated on keratinocytes. Detailed study on the transmission pathway of these receptors suggested a plausible explanation for the action of FROP-s and RROP-s at the level of the message-transmission between skin cells and extracellular messages and also at the level of cell-matrix interactions. The nuclear penetration of FROP-s, about 8-times more intense (as estimated by the measurement of fluorescence intensity) suggests a direct action on gene-expression and **regulation.** Further studies are indicated in order to fully elucidate the mechanisms of the above summarised remarkable "anti-aging" properties of RROP-s and FROP-s.

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applied to the human skin of a team of voluntary women encompassing all age-groups, the efficiency of FROP-containing preparation could be confirmed using indentometry and computerised evaluation of skin micro-relief, as well as evaluation of periorbital wrinkles. It appears therefore that these preparations correspond to all the requirements of active anti-aging principles. Skin aging became an important issue of our rapidly aging society. 20 to 35% of the general population in all advanced and in most advancing countries is above 65 years. The most rapidly increasing fraction of this population are the very old (centenarians). This fact creates an important market for anti-aging skin products addressing an increasingly exigent population. Modern research carried out in some laboratories is taking up this challenge with increasing efficiency. Before describing the results obtained in our laboratory along these lines of investigation, we have to discuss in some detail the basic cellular and molecular mechanisms involved in skin aging. In order to define the most significant tests for screening studies, the first important issue is to define the mechanisms which underly skin aging. Aging of tissues is a complex process which involves the aging of cells, of extracellular matrix (ECM) and of cell-matrix interactions. These interactions are mediated by receptors as the integrins or the elastin-laminine-receptor (ELR). Both types of receptors (as well as some others) are involved in the aging process. Cell-aging, as studied in cell cultures, follows the principles elaborated by L. Hayflick and is explained essentially by the telomer-**telomerase**-centered mechanisms. Cell aging affects cell divisions both for keratinocytes and fibroblasts which is progressively **slowing** down with age. As shown however by the **slow** but efficient wound healing of elderly subjects, it remains sufficient to close surgical or accidental wounds. An other important aspect of cell aging is the progressive modification of the 'program' of the biosynthesis of ECM components. This is an important aspect of skin aging because of the rich ECM of dermis. Loss of dermal tissue measured as the skin thickness of biopsy specimens from sun-protected sites gave an average estimate of 7% of the original skin thickness (extrapolated to birth) lost every 10 years. This leaves less than the one third of the original skin thickness at about 90 - 100 years. This loss of skin tissue is the combined result of cell loss and mainly of **reduced** cell-biosynthetic activity, as well as of increased proteolytic activity. Age-dependent loss of collagen is the result of the combined effect of decreased biosynthesis and increased degradation, as the result of the age-dependent increase of the local production of matrix degrading enzymes. As shown on Fig.2., elastase-type endopeptidase activity is steadily increasing with chronological age and also during in vitro aging, with increasing cell passages as shown with human skin fibroblasts. Decreasing matrix biosynthetic activity combined with increasing matrix degradation are the two essential ingredients of skin aging. Besides proteolytic enzymes, reactive oxygen species comprising free radicals as hydroxyl radical, superoxide and hydrogen peroxide represent another important source of skin degrading agents. ROS-production is both an intrinsic, cell-dependent process and also a photochemically, UV-induced mechanism. It was shown however, that UVA-induced free radical production was much more important than UVB-induced production, is maximal at the skin surface and decreases rapidly towards the dermis. The metabolic generation of ROS is however cell-dependent, essentially of mitochondrial origin, and was shown to increase with age, together with a decrease of the cellular scavenging activity. We could show that hyaluronan, one of the most important glycosaminoglycan components of skin, is highly sensitive to free radical degradation. This reaction could be used for the quantitative determination of free radical generation. Hyaluronan is produced both in

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L123 ANSWER 47 OF 48 KOSMET COPYRIGHT 2004 IFSCC on STN
ACCESSION NUMBER: 23564 KOSMET
FILE SEGMENT: miscellaneous
TITLE: BENCH & BEYOND: A DOUBLE TAKE ON ANTI-AGING
AUTHOR: BREWSTER B (C/O EDITOR, COSMETICS & TOILETRIES, 362 SOUTH SCHMALE ROAD, CAROL STREAM, IL 60188-2787, USA)
SOURCE: COSMET TOILETRIES, 2001, 116, 5, 6-10, 7 REFS
DOCUMENT TYPE: General review
LANGUAGE: English
AB Recent skin-care launches suggest two mechanisms and two attitudes for providing anti-aging treatment to human skin. The two mechanisms work at the cellular level are **telomerase** enzyme and gerontogenes modulation by kinetin (N6-furfuryladenine). The two attitudes focus on the clock (by **reducing** the signs of aging)
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L123 ANSWER 48 OF 48 KOSMET COPYRIGHT 2004 IFSCC on STN
ACCESSION NUMBER: 14930 KOSMET
FILE SEGMENT: scientific, technical
TITLE: TELOMERASE ACTIVITY CONCENTRATES IN THE MITOTICALLY ACTIVE SEGMENTS OF HUMAN HAIR FOLLICLES
AUTHOR: RAMIREZ R D (DEPARTMENTS OF CELL BIOLOGY AND NEUROSCIENCE AND DERMATOLOGY, THE UNIVERSITY OF TEXAS SOUTHWESTERN MEDICAL CENTER AT DALLAS, DALLAS, TEXAS, USA); WRIGHT W E; SHAY J W; STAN T R
SOURCE: J INVEST DERMATOL, 1997, 108 (1), 113 -117, 35 REFS
DOCUMENT TYPE: Journal
LANGUAGE: English
AB **Telomerase** is a ribonucleoprotein enzyme capable of adding hexanucleotide repeats onto the ends of linear chromosomal DNA Whereas normal somatic cells with a limited replicative capacity fail to express **telomerase** activity, most immortal eukaryotic cells do. Cells of renewal tissues (e.g., skin, intestine, blood) require an extensive proliferative capacity. Some cells in such renewal tissues also express **telomerase** activity, most likely to **prevent** rapid erosion of their telomeres during cell proliferation. In this study, we measured the levels of **telomerase** in dissected compartments of the human hair follicles : hair shaft, gland-containing fragment, upper intermediate fragment (where it is thought undifferentiated stem cells resides), lower intermediated fragment, and in the bulb-containing fragment (an area whith high mitotic activity containing a more differentiated pool of keratinocytes). In anagen follicles, high levels of tolerase activity were found almost exclusively in the bulb-containing fragment of the follicles, with low levels of **telomerase** in the bulge area (intermediate fragments) and gland-containing fragment. In comparison, catagen follicles had low levels of **telomerase** activity in the bulb-containing fragments as well as in other compartments. Such observations indicate that, in anagen hair follicles, the fragments containing cells actively dividing (e.g., transient

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